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Listerian Oration.[1]

THE LIVER AND THE PORTAL CIRCULATION.

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THANK you for the honour of inviting me to address you this evening—I am appreciative of the significance of my *appearance here because I have read, and marked,* the names of my eminent predecessors.

Thirty-five and a half years ago my father delivered the Listerian Oration here in this city, and this fact adds greatly to my own pleasure in being here. He chose the *title "The Clinical Interpretation of Blood Pressure* Readings" (Blackburn, 1923); he spoke especially from his clinical experience, and his theme was aptly stated:

> . . . I fancy the medical attendant is a little inclined to stand in awe of his instrument and to base his prognosis and treatment too lavishly on its readings, forgetting that his sphygmomanometer is primarily a means by which he can record in figures observations *that his predecessors made with their fingers, assessing* their true value after careful investigation of heart, blood vessels and kidneys.

[1] Delivered at a meeting of the South Australian Branch of the British Medical Association on April 30, 1959.

The interpretation is more important than the reading; the whole is greater than the sum of the parts.

I have had a special interest in liver disease and the portal circulation for many years, and have been fortunate enough to have had opportunities to investigate, and to be *associated with young men investigating, the portal* circulation in patients with liver disease at the Clinical Research Unit of the Royal Prince Alfred Hospital and the Department of Medicine in the University of Sydney. I also have a little clinical experience in this field, so it *is appropriate that I discuss the "Liver and the Portal* Circulation" with you this evening.

Lister's major contributions to medicine rested upon his application of his interpretation of data primarily produced by Pasteur. His interpretations led to the better *management of his and our patients, and made our* investigations possible. I hope some benefit to patients will derive from the interpretation of data obtained from the study of patients with liver disease and of changes in their portal circulation.

It is necessary for me to provide some anatomical, physical and physiological background before I discuss the changes in disease and attempt to formulate an hypothesis, but I adhere to the view expressed by my father that the interpretation is of the greatest importance, and so will spend more time on interpretations than on detailed data.

Anatomical Considerations.

Developmental Anatomy.

The primitive alimentary canal differentiates into three *parts, the foregut, the midgut and the hindgut,* and these are also functional divisions.

The foregut is represented by the pharynx, respiratory tract, œsophagus, stomach, liver, pancreas and spleen. *In* an "alimentary" sense it is concerned with the preparation *of food for digestion and absorption; its artery of supply* is chiefly the cœliac axis, and its venous drainage mainly to the portal vein. The developmental connexion between the lungs and *the intraabdominal part of the foregut* derivatives is relevant to my discussion.

The midgut represents the chief part of the primitive alimentary canal, and develops into the digesting and absorbing part of the alimentary tract down to the splenic flexure; *its arterial supply is the superior mesenteric* artery, and venous drainage is by the left vitelline vein, *later to become* the superior mesenteric vein draining into the portal vein.

The hindgut extends from the splenic flexure to the anus, and *the bladder and urethra develop from its lower* end. This part of the gut is excretory; it is supplied by *the inferior mesenteric artery,* and drained by the inferior mesenteric vein, terminating in the portal venous system.

The hepatic bud develops just behind the sinus venosus, and grows *into the vitelline veins which form the small* venous channels in the liver. The hepatic veins are collecting or efferent vessels; they develop from the right vitelline vein and connect with the sinus venosus. *The* portal venules are distributing or afferent channels; they *develop from the left vitelline vein,* and connect with the left umbilical vein in the transverse fissure of the liver to form the portal sinus. A direct channel, the ductus venosus, develops *between the portal sinus and the inferior* vena cava. Immediately after birth the umbilical vein *atrophies,* and muscular contraction of the ductus venosus leads to its permanent closure.

I wish to draw attention to the close developmental relationship of the liver to the *rest of the alimentary tract,* and especially to the derivatives of the foregut, including *the lungs. There is a close* developmental relationship between the vasculature of the liver, the lungs and the rest of the foregut.

Anatomy of Splanchnic Vasculature.

Macroscopically, we are chiefly concerned with the branches of the cœliac axis and the portal vein tributaries.

The major arteries are the hepatic, the gastric, the splenic and the *mesenteric, and they freely anastomose* with their neighbours, forming a splanchnic arterial bed. *This is of importance* when one is considering surgical ligation of arteries to decrease portal vein inflow.

The splanchnic venous drainage is to the portal vein, which is formed by the junction of the left gastric or coronary vein, the splenic vein and the superior mesenteric *veins, though many variations occur,* as would be expected from the complicated embryology.

Reference must be made to the connexions with the systemic venous system by the œsophageal, perio-œsophageal and vagal veins, by the plexuses around the left kidney and the adrenal gland, and by the diaphragmatic plexus, along the falciform ligament to the anterior abdominal wall and through the hæmorrhoidal plexuses. These are of great importance in disease states.

The microscopic arterio-venous anastomoses in the splanchnic circulation are of great functional importance and interest. Those in the intestinal villi are well recognized. The splenic circulation is complex, but there are pathways equivalent to arterio-venous anastomoses, and there are huge shunts in the stomach which allow glass spheres 140μ in diameter to pass (Walder, 1950). Arterio-portal venous shunts are easily demonstrated in patients with cirrhosis, and, indeed, retrograde flow has been described in the portal vein at laparotomy.

The hepatic arterial supply is from the hepatic artery, the hepatic arterial-venous supply is by the portal vein, in which the blood has a *high oxygen saturation, and the* hepatic venous drainage is via the hepatic veins to the *inferior vena cava.* The hepatic artery (or arteries) arises from the cœliac axis as a rule, and is *relatively* small, whilst the portal vein is large—about 2 cm. in *diameter—and has a relatively thick muscular wall for a* vein.

The essence of the hepatic vasculature is the sinusoid, into which both the portal venules and the *terminal twigs* of the hepatic artery enter at the "periphery" of the *lobule, and which drains into a central vein* leading to the hepatic veins. The sinusoids form a huge sinusoidal bed, which is not dissimilar to a sponge. They are in no sense end vessels, but *freely intercommunicate with one another.* If a given hepatic venule is completely obstructed, the *pressure* in the draining sinusoids will be that of the sinusoidal bed. Since the blood from the *portal vein* drains into the sinusoids, this is the basis for the estima*tion of portal venous pressure in man* by hepatic vein catheterization (Myers *et alii,* 1951). It has been demonstrated in man (Paton *et alii,* 1953) that the pressures recorded *in this circumstance closely approximate portal* venous pressures, provided that there is no presinusoidal *obstruction.* The connexion between the arterial and portal venous vessels is most important, because *it constitutes* an arterio-venous anastomosis which is often opened up in *liver disease.*

Physical Considerations: Some Physical Laws Governing Flow in Tubes.

These physical laws are mentioned because their *appreciation allows greater understanding of the changes* in the portal circulation in disease, and of some principles *of management.*

Poiseuill's law states that the pressure difference of a fluid flowing through a tube is a function of the product *of the amount flowing and the resistance to flow; the flow* depends on resistance and pressure. The pressure difference between the ends of a tube through which a liquid is flowing depends directly on the *viscosity of the fluid,* the length of the tube and the quantity flowing, and varies *inversely with the fourth power of the radius.* An important aspect of this law, especially in my present context, is that the resistance to flow depends on the *viscosity of the fluid and on the geometry of the tube.*

It is important to stress that pressure, flow and resist*ance are so interdependent that flow increase* may be as effective as resistance increase in producing a pressure rise—think of the fully turned on tap versus a little *dribble. Viscosity is important, and a consideration of* the geometry of a vessel provides one reason why a shunt between the splenic vein and renal vein can successfully decompress portal vein hypertension, even *though it adds* relatively little to the cross-sectional area of the venous *tree. A shunt through a single large short vein has a much* lower resistance than that through a series of small long *veins of much larger total cross-sectional area.*

Bernoulli's law is most simply expressed by stating that the total energy of flow of a liquid through a closed tube is constant and equals the kinetic energy plus the potential energy. This means that the slower the flow, because of the local dilatation of the tube, the greater the static pressure on the walls. This is one factor in the production of the so-called post-stenotic dilatation.

Laplace's law has a similar application, in that it states that the tension on the wall of a tube equals the product of the radius of the tube and the radial pressure on the wall. Large vessels therefore must have strong walls. The portal vein must have adequate walls; capillaries and venules need the thinnest of walls.

Laplace's law and Bernoulli's law explain the tendency for a swelling of a vein, or an arterial aneurysm, to extend continuously once it starts. Large varices mean large veins with weak walls as much as high-pressure "blow-outs". We all know that the initial distension is the

really hard part of blowing up a child's balloon—the bigger it gets, the easier it is to blow up.

Physiological Considerations.

Regional Splanchnic Flow.

The inflow to the portal vein is principally from the spleen, the stomach and the small intestine, and the flows from these viscera are of great importance.

The circulation within the spleen seems to be sluggish, and often is, but there is a large artery and a large vein. It has been said that the splenic vein can contribute 40% of the portal venous flow, and it may be more when there is gross splenomegaly in cirrhosis. Small doses of adrenaline can double the splenic blood flow.

In the gastric submucosa the large arterio-venous anastomoses can open in various circumstances; for example, if there is obstruction to the capillary bed (Walden, 1952). Burton (1952) relates their opening and closing to critical pressures; open with high pressure, closed with low. Grant (1952) regarded the anastomoses as the same as those in the rabbit ear, or those in the hand. More recently Peters and Womack (1958) have shown that these anastomoses may open up, as a shunt, after the injection of adrenaline, but that after the injection of histamine, or during active gastric secretion, they are closed, and capillary flow is dominant. It is of some interest that Peters and Womack (1958) demonstrated a rise in portal vein pressure and oxygen saturation when the anastomoses were made patent by administering adrenaline.

Arterio-venous anastomoses in the intestinal villi are considered to have reactions quite comparable with those of the hand (Grant, 1952).

In summary, there is evidence that the anatomical arrangements in the splanchnic vascular bed, especially with regard to the presence of arterio-venous anastomoses, are functionally significant in affecting portal vein inflow. Their functional significance in digestion, exercise, stress and hæmorrhage needs little emphasis, and their relevance to liver disease will be referred to later.

Portal Blood Flow.

Little emphasis has been placed on blood flow in the past, and most attention has been given to portal obstruction, intrahepatic or extrahepatic. As was mentioned earlier, the splanchnic inflow is as important as resistance to flow.

Portal venous flow obviously depends particularly upon arterial input to the splanchnic bed, upon portal and hepatic resistance to flow, and upon the portal venous pressure. In this connexion the data of Peters and Womack (1958) are of interest, because the rise in portal vein pressure after the administration of adrenaline could be due to increased gastric flow through shunts, to increased resistance to flow from the portal vein, or to a combination of both factors.

Portal venous flow is about 80% of the estimated hepatic blood flow in the normal person, and this represents, in the main, the sum of the gastric, splenic and mesenteric flows, since practically all splanchnic blood goes through the liver.

Hepatic Blood Flow.

In normal man the average estimated hepatic blood flow is about 1500 ml. per minute (Bradley *et alii*, 1952), and this equals about one-fifth of the cardiac output. We have used bromsulphthalein, radioactive colloidal gold, and radioactive colloidal chromic phosphate clearance methods, with hepatic vein catheterization, and have obtained comparable results, though the methods are not simple.

Hepatic blood flow includes hepatic arterial flow and portal venous flow, which normally appear to provide 20% to 30% and 70% to 80% of the total flow respectively. If the portal venous flow is reduced by, say, ligation of the vein, arterial inflow immediately increases owing to a decreased resistance to flow in the liver (Sancetta, 1953). However, changes in arterial blood pressure from 60 to 160 mm. of mercury have little effect on total hepatic blood flow in the normal person, on account of "intrinsic regulation of liver blood flow" (Grayson, 1954a). Grayson also suggests an undetermined role for the spleen in this phenomenon.

Hepatic blood flow is affected by many factors, of which the following are important:

1. The resistance to blood flow (R), which depends on the viscosity of the blood and on the geometry of the blood vessels, their length and especially their radius. This last factor would include: (i) changes in the hepatic venous resistance—that is, post-hepatic factors; (ii) changes in the hepatic arteriolar and venular, the sinusoidal, and the presinusoidal resistance—intrahepatic factors; and (iii) changes in the hepatic arterial and portal venous resistances to flow—pre-hepatic factors. Special importance must be given to vasomotor effects and mechanical obstruction.

2. The quantity of blood entering the hepatic vasculature (Q). This includes the total and splanchnic blood volume, and the regional splanchnic blood flow, with special reference to arterio-venous anastomoses.

3. The blood pressure (P), both arterial (in patients with cirrhosis of the liver) and portal venous.

The particular role of these factors, R, Q and P, in liver disease varies from patient to patient; but they provide rational bases for such therapeutic measures as rest for patients with hepatitis, splenectomy and gastric devascularization for bleeding varices, and phlebotomy for portal hypertension.

Hepatic vasomotor adjustment is the key factor determining hepatic blood flow in ordinary circumstances, but there are differences of opinion regarding the precise effect of the therapeutic and physiological agents on the human hepatic vasculature.

Splanchnic Blood Volume.

The volume of blood in the splanchnic bed in man can be estimated, and the mean value as determined by Bradley and his colleagues (1953) is 1025 ml. (S.D. ± 300 ml.), which is about one-fifth of the blood volume. It is affected by a variety of factors, which include the total blood volume, the size and tone of the blood vessels and the inflow-outflow relations.

It has been shown that a marked decline in splanchnic blood volume occurs after blood loss, but without splanchnic arterial vaso-constriction, so that hepatic blood flow is maintained: venous constriction occurs. An increased flow through splanchnic arterio-venous anastomoses will increase portal venous inflow, and a decreased capillary flow combined with a decreased calibre of the portal venules will decrease the capacity of the splanchnic bed, so that rate of flow and pressure in the portal vein will increase and maintain hepatic blood supply. This series of events appears to occur after hæmorrhage, but, in exercise, splanchnic flow decreases as a result of vaso-constriction (Bradley *et alii*, 1953).

These findings are important because they indicate that exercise should not tend to produce hæmorrhage in the patient with portal hypertension on this account alone. Furthermore, the relationship of the splanchnic blood volume to the general blood volume provides us with a physiological reason for making every effort to avoid an increased blood volume—indeed, for attempting slightly to lower it—in patients with œsophageal varices.

Changes in Disease.

Effects of Portal Venous Obstruction and the Development of Œsophageal Varices.

In man and in the dog, ligation of the portal vein is not followed by persistent features of portal hypertension; that is, œsophageal varices and distended veins. There is an immediate increased arterial flow to the liver, and transient venous effects may be observed, but in the dog and in the monkey the portal vein pressure usually (Child, 1954) returns to normal. Ligation of the portal

vein in "normal" man is not usually followed by clinical portal hypertension.

Portal vein obstruction developing in infancy and childhood, however, is commonly associated with splenomegaly and often with œsophageal varices. Portal vein obstruction added to hypoproteinæmia, or fluid retention, will cause ascites and, indeed, a few cases of ascites occurring in extrahepatic portal venous obstruction have been described (Baggenstoss and Wollaeger, 1956).

Obstruction should cause significant increase in resistance to flow and a rise in portal venous pressure unless flow is reduced. This increase in pressure may suffice to ensure blood flow through the portal vein, but if there is a disorder of the vein walls, or if the pressure is very high, there may be dilatation of veins and increased splanchnic blood volume.

In patients with portal vein obstruction and liver disease, the collateral circulation commonly becomes more patent, especially around the œsophagus, the vagus nerves, the left kidney and adrenal, the ligamentum teres and the umbilicus. The development of collateral venous channels tends to limit further increase in portal pressure. Indeed, there may be a decrease in portal venous pressure, and the patient may "recover", or reach a static condition when collateral circulation becomes fully developed: neither progression nor regression will occur. We have seen patients who appear to demonstrate this sequence of events: for example, a girl with portal hypertension and œsophageal varices developed a large vein in her abdominal wall after cardio-œsophageal resection, but the distended vein disappeared in a few weeks without return of varices. Presumably better channels for venous drainage had developed.

There is, perhaps, no need to interfere surgically with such "static" patients unless they have unexplained bleeding, not, for example, bleeding occurring after ingestion of aspirin or dietetic indiscretions. We are studying the cases of several children with extrahepatic portal obstruction and œsophageal varices who seldom have bleeding and do not appear to have progressive disease. Surgery is not recommended now.

Other possible effects of collateral circulation, as listed by Sherlock (1958), are: decreased spleen size, small liver, encephalopathy, septicæmia, and decreased portal blood flow into the liver.

The collateral circulation at the cardio-œsophageal region is most important, because varices often do bleed. The rest of the collateral circulation does not seem to have clinically significant baneful effects, and is to be regarded as beneficial. Childs (1954) has said that portal hypertension is significant when œsophageal varices are demonstrable on X-ray examination after a barium swallow, and we find this to be a good clinical rule. He reported that bleeding occurred in his patients with œsophageal varices if their portal pressure was 300 mm. of saline (22 mm. of mercury) or over, but if the pressure was below this, the risk of spontaneous bleeding was small.

Œsophageal varices usually appear to extend upwards from the cardio-œsophageal region, but sometimes they occur in a localized area well above this region. These venous channels become much smaller before they enter larger veins, ultimately to end in the azygos, the bronchial or the pulmonary veins, the vena cava and the left auricle, so we cannot assume that there is much blood flow in them.

Some varices do appear to carry a significant amount of blood, because we have seen grossly dilated hemi-azygos and azygos veins at operation and in X-ray films. Two of our patients with extrahepatic portal vein obstruction developed obvious abdominal wall veins, with downward flow, shortly after resection of the œsophago-gastric region for bleeding varices. We have seen practically all the contrast medium injected into the spleen, during splenoportography, drain upwards through veins in the œsophageal region.

The strict interpretation of œsophageal varices is that there is a dilatation of the submucous veins. They occur in a variety of clinical circumstances, such as cirrhosis of the liver, extrahepatic portal venous obstruction, congestive cardiac failure and old age. They can be due to several causes, for instance, portal hypertension, increased splanchnic blood volume, abnormalities in the vein walls, and increased splanchnic blood flow. Clinically, it is important to consider the possible cause of varices, and to avoid an immediate assumption of portal hypertension.

Varices are often present in patients with portal hypertension, and in this circumstance the mechanism is not hard to understand if we think of Bernoulli's and Laplace's laws, and allow for some weakness of the vein walls. Local venous changes must influence the development of varices, and, similarly, the extent to which the collateral circulation develops depends on a variety of circumstances; for example, dilated vessels around the diaphragm and œsophageal varices are common in congestive cardiac failure (Palmer and Buik, 1955).

In some patients with liver disease we have estimated the portal pressure and found it raised, but no varices could be demonstrated. Palmer has described the appearance and disappearance of varices under static clinical conditions. Nevertheless, varices do tend to bleed, and portal hypertension is clinically significant when it is associated with varices.

We have been led into error by the presence of œsophageal varices in a patient with cirrhosis, but without demonstrable portal hypertension as measured in many veins at laparotomy. He had normal pressures in a number of radicles of his portal vein on hepatic vein catheterization, obvious œsophageal varices and inactive cirrhosis at laparotomy. There may be little flow in his varices, and they may have persisted after developing years earlier, though such a contention is not completely supported by an unspectacular response to injection.

There are factors other than portal hypertension in the development of œsophageal varices, and important additional ones are: (i) portal inflow, as affected by increased arterial inflow from the splanchnic bed; (ii) the state of the vein walls, which appear to be less able to withstand raised pressure in patients with cirrhosis than in normal subjects; (iii) the age of the patient, since varices are not rare in the aged (Weinberg, 1949).

It has been noted that evidence of increased collateral circulation, apart from varices, is rare in our patients with extrahepatic portal obstruction and without liver disease, but we have seen transient abdominal wall veins twice, which does not suggest that obliteration of the collateral vessels is the reason for this.

Changes in the Splanchnic Circulation.

Changes in the splanchnic circulation, and especially in the portal circulation, are common in liver disease, and the characteristic clinical change is the development of œsophageal varices. It is also common for cirrhotic patients to present with bleeding from acute gastric erosions, even when they are not alcoholics; and peptic ulcer is common. In most large series of cases with cirrhosis and upper gastro-intestinal tract bleeding, ruptured œsophageal varices account for only about one-third.

We find the ingestion of aspirin a common antecedent to bleeding in our cirrhotics; it has vied with alcohol for the ætiological crown. Alcoholics often take aspirin for breakfast.

Venous obstruction occurs in cavernous transformation of the portal vein, and it may also occur within the liver itself, in the presinusoidal vessels. Both of these lesions can cause portal hypertension. In Laennec's cirrhosis, necrosis of liver cells results in architectural collapse; and irregular regeneration and fibrosis result in distortion of architecture and venous obstruction. The net result may be obstruction to flow which is post-sinusoidal, hepatic or central venular, sinusoidal or presinusoidal or portal venular. There may also be decreased resistance

to flow in other areas, since another effect of collapse of the lobule is shortening of the pathway from portal venule to hepatic venule. Laennec's cirrhosis often results in portal hypertension and, more commonly than pure portal venous obstruction, in ascites.

As was shown very many years ago (Herrick, 1907), there is an increased contribution from the hepatic artery to the sinusoid in cirrhosis. The portal pressure rises much more in the cirrhotic liver than in the normal when the arterial pressure is raised a given amount. There are increased functioning arterio-venous anastomoses in the cirrhotic lever. Reversed flow has been described in the portal vein in cirrhosis at operation, hepatic arterial flow in the liver was seen to be leaving by the portal vein to reach the collateral vessels. Ligation of portion of the hepatic artery in man has been shown to lower portal pressure.

There appears to be an increased portal inflow via the splenic vein, since the spleen is commonly large in cirrhosis, and, in this circumstance, the splenic artery is usually increased in radius, though the arterial pressure is not raised. Splenectomy alone will often lower the portal pressure in cirrhosis.

There are several surgical procedures that are beneficial to patients with bleeding œsophageal varices: construction of decompressing portal-systemic shunts, excision of the varicose area, injection of varices, excision of the stomach and varicose area and ligation of much of the blood supply to the stomach. We have aimed to lower pressure (P), to decrease the inflow (Q), and to remove the bleeding area. Cases in which the construction of shunts has been performed (by Mr. F. H. Mills and Professor J. Loewenthal) have been the most successful, but benefit has also occurred in some patients without active cirrhosis from the injection of varices. "Devascularization" of the stomach has been successful upon occasion, but failed completely in a girl with actively progressive hepatitis. Consistently good results should not be expected, since there is a splanchnic bed rather than a separate regional blood supply.

We have now had the opportunity to observe the effect of controlling the viscosity of the blood and, perhaps, the blood volume.

On the basis that pressure and resistance vary directly with viscosity, and that several patients stated that bleeding occurred as soon as their blood volume was "up to normal", we prescribed phlebotomy for a girl, aged 12 years, with portal hypertension and œsophageal varices that bled. Her spleno-renal anastomosis had failed, but her liver function was normal. She had her longest period of freedom from bleeding since her first episide whilst her local doctor kept her hæmoglobin level down to 9 to 10 grammes per 100 ml. When this rose to 14 grammes per 100 ml., bleeding promptly occurred again.

There is evidence that the following changes may occui in the splanchnic circulation in patients with cirrhosis of the liver: (i) venous obstruction, intrahepatic and extrahepatic; (ii) increased collateral circulation; (iii) increased inflow through splenic and gastro-duodenal veins; (iv) opening up of arterio-venous anastomoses in the stomach, small intestine and liver.

Changes in the Systemic Circulation.

The changes which may occur in the systemic circulation of patients with cirrhosis of the liver are well known: spider nævi on the skin surface, palmar erythema, hyperkinetic circulation, a high cardiac output, and an increased blood volume. Finger clubbing, an enlarged heart, ankle œdema without hypoproteinæmia and a raised venous pressure gradient have all been seen in our patients. We have found a slight increase in forearm blood flow and an increased blood flow in the hand in some of our cirrhotic patients.

It is notable that systemic arterial hypertension is seldom seen in patients with cirrhosis of the liver.

These findings may be related to an increased blood volume, to more functioning peripheral arterio-venous anastomoses and a decreased peripheral resistance, and to changes in the vessels themselves which result in spider nævi and palmar erythema. The cardiac output has been reported to correlate with the presence of increased collateral circulation in cirrhosis (Murray *et alii*, 1958) and with plasma volume (Claypool *et alii*, 1957).

Though some of the systemic vascular phenomena can be explained as being secondary to a large blood volume, others cannot—for example, the absence of arterial hypertension and the changes in the vessels themselves. It can be argued that an increased blood volume is secondary to peripheral vasodilatation rather than primary. Sherlock *et alii* (1958) have suggested that the systemic changes are due to the appearance of some vasodilator substance appearing in the peripheral circulation via the collateral circulation.

Changes in the Pulmonary Circulation.

Over the past few years we have studied arterial oxygen saturations in patients with liver disease and have found that in many normal saturation is not reached. In some, full lung function studies did not reveal any significant lung disease, and the breathing of 100% oxygen did not restore the arterial oxygen saturation to normal. Similar findings have been reported by Snell (1935-1936), by Wilson *et alii* (1953) and by Sherlock *et alii* (1958). More recently, Rydell *et alii* (1956) described a child with cirrhosis and cyanosis in whose lungs arterio-venous shunts were demonstrated at autopsy.

We have also been interested in the frequency with which our cirrhotic patients have arterial oxygen unsaturation due to the venous admixture effects of chronic lung disease, represented by bronchitis and emphysema.

There is evidence that the venous shunt, not due to lung disease, is intrapulmonary rather than portal-pulmonary via the œsophageal-vagal-left auricular route, although shunting by this route has been demonstrated at autopsy by injection studies in cirrhosis and after operations for extrahepatic portal obstruction. More than one patient has had oxygen saturations of 85% or less, and, since the portal venous blood is considered to be about 60% saturated, an amount of blood greater than the estimated splanchnic flow would have to flow into the left side of the heart to account for the arterial unsaturation we have found. Indeed, if there is any degree of arterio-venous shunting in the splanchnic bed, our estimate of the amount shunted is too conservative.

Whilst arterial oxygen unsaturation in patients with cirrhosis may be due to chronic lung disease, to portal-left auricular shunting, and to pulmonary arterio-venous shunting, I regard the pulmonary shunt as of the greatest significance, even though it is difficult to demonstrate uniquivocally except in the minority of patients.

Conclusions.

I suggest that the changes in the splanchnic circulation do not greatly differ from those in the other major circulation areas in patients with cirrhosis, except for venous obstruction. Identical changes are not seen in all patients, and the pathological emphasis may be more in one area than in another.

Vasodilatation, arterio-venous shunting and increased blood volume appear to be the characteristic functional changes in the peripheral circulation in patients with cirrhosis of the liver, and in some other forms of liver disease. An increased cardiac output is common. Structural changes in the blood vessels are easily recognized as "spider nævi".

Arterio-venous shunting can be demonstrated, we believe, in the lungs of some patients with cirrhosis, and the resulting arterial unsaturation may be a factor in the peripheral vasodilatation and hypotension, but we have no solid data to support this.

There is reason to suppose, from experiment, that the smaller blood vessels of the splanchnic bed behave like other small blood vessels, and that the changes that

occur in the peripheral and pulmonary circulations may be expected to occur in the splanchnic bed. This is specially so if there is some circulating agent causing the functional changes.

The systemic, the pulmonary and the splanchnic circulations appear to be affected similarly, and venous obstruction—postsinusoidal, presinusoidal or extrahepatic—contributes greatly to the clinical phenomena of the changes in the portal circulation.

It is suggested that the changes in the portal venous system in patients with cirrhosis of the liver may be attributed to venous obstruction, increased arterial inflow, increased splanchnic blood volume, and changes in the walls of the small blood vessels and veins. The increased arterial inflow appears to be due to opened arterio-venous anastomoses in the liver, stomach and intestine, and to increased spleen flow, and these anastomoses can enormously increase local flows. An increased splanchnic blood volume may be due to increased inflow, increased hepatic resistance and changes in the vessel walls themselves. Œsophageal varices may be due to a combination of effects operating through simple physical laws: these are slow flow, increased pressure and decreased strength of the vessels' walls.

The precise cause of the changes in the circulation in patients with cirrhosis is not clear, but it can be suggested that there is evidence that a single fundamental mechanism is operating.

Sir Isaac Newton's first two "Rules of Reasoning in Philosophy" (1686) are: (i) "we are to admit no more causes of natural things than such as are both true and sufficient to explain their appearance"; (ii) "therefore to the same natural effects we must, as far as possible, assign the same causes". The application of these leads me to the following hypothesis.

In many patients with cirrhosis of the liver some circulating substance so affects the walls of smaller blood vessels that vasodilatation and opening of arterio-venous anastomoses occur in the peripheral, splanchnic and pulmonary circulations. These changes are responsible for the changes observed in the peripheral and in the pulmonary circulation, but additional factors also affect the portal circulation. In the portal circulation the effect of obstruction to flow accentuates the tendency to raised venous pressure, induced by the changes in the splanchnic circulation as a whole, and this, together with a change in the vein walls, leads to the development of large dilated venous channels, some of which are œsophageal varices.

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DOES PRESSURE ON NERVES CAUSE PAIN?

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What is needed at this moment of the history of neurology is the synthesis of facts into an organized whole. This cannot be done without doing injustice to many a valuable observation.

WALTHER RIESE: "Principles of Neurology", 1950.

A GREAT DEAL of modern medical and surgical practice is based on the supposition that most unexplained pains are due to pressure. When the cause of a pain is unknown, anyone who suggests a likely mechanism which is capable of generating pressure or tension on nerves will probably have his explanation accepted.

Thus the pain which shoots down the arm is assumed to come from pressure on cervical roots or on the brachial plexus. Sciatic pain, too, is attributed to pressure on nerve roots. Post-traumatic headache is said to come from tension on meningeal adhesions, and migraine from the stretching of nerve fibres in dilated intracranial arteries. Hurst (1911) said that colic was due to pressure on fibrils in the contracting visceral walls. The tension hypothesis of inflammatory pain has been well stated by Kinsella (1953). The pain which accompanies malignant growths is due to pressure by tumour cells on pain nerves. Many further examples could be given.

Though the word pressure is applied equally to all these cases, they can be divided into three unrelated mechanisms: (i) Constriction of the axis cylinders of a nerve root by an intervertebral disk. (ii) Compression of pain plexus fibrils by contracting smooth muscle. This does not resemble the first mechanism. (iii) Both of these pressures are entirely different from the infinitesimal tension which develops in the tissues around a furuncle; this can be called pressure only by a stretch of the imagination.

There is even less tension in the edge of a growing tumour.

Other objections to the pressure theory come to the mind. In œdema and giant urticaria, the tension may be greater than in inflammation, and yet it is painless. The extreme pressure of clamping the bowel wall does not cause pain. Tumours of nerve trunks are frequently painless. Yet the theory is supported by many daily observations. The pain of digital cellulitis or of migraine throbs with the pulse; occlusion of the carotid artery temporarily relieves migraine. Incision of an abscess relieves pain at once through lessening of tension. But in most of these painful conditions the tissues have already been hyperalgesic.

Deep Pain and Pressure Sense.

Pressure sense and deep pain are closely related. The skin is not concerned with them, and the receptors of both are located in the deep fascia and periosteum (Weddell and Harpman, 1940; Kelly, 1945).

Observation 1.—(*a*) I anæsthetized a patch of my tibial periosteum. When I pressed it with my thumb through normally sensitive skin, I felt no pressure sense. A blow with a hammer was painless. (*b*) When a patch of skin over the tibia was anæsthetized but the periosteum was not, the pressure sense was the same through anæsthetized as through normal skin. A blow with a hammer caused pain of equal intensity on both spots.

In different skeletal tissues, however, different pressures are required to elicit pain. Though pressure sense and pain sense are related, they are distinct both anatomically and physiologically.

Observation 2.—(*a*) If X is the thumb pressure on the belly of my biceps muscle which induces pain, 2X is required on the tibial periosteum and half-X in the testis and on certain spots of greater intensity near musculotendinous junctions. (*b*) The tuber ischii and the pads of the thumb and heel have pressure sense, but 5X is required to induce pain. The periosteum of the tooth socket painlessly records great pressures. (*c*) On the tooth pulp, conjunctiva, nasal mucosa and in hyperalgesic tissues, the lightest touch without pressure may cause pain.

Pain from Sensory Trunks or Roots.

It is generally thought that nerve root pain is due to pressure on the sensory axons themselves, rather than on the nervi nervorum in the sheath. This mechanism is unrelated to that by which tension in the tissues stimulates the pain plexus. Sensory nerve trunks are tender to firm pressure, but not more tender than many other tissues which contain the pain plexus but no nerve trunks. Waller (1862), Head (1896) and Sherrington (1900) recognized that damage to axis cylinders often did not cause pain to radiate into their peripheral fields.

Observation 3.—When I struck my ulnar nerve on the edge of the table, I felt severe pain at the site of the blow (the elbow) for five minutes. It radiated half-way down the forearm, but not to the hand. In the fourth and fifth fingers I felt numbness, then for thirty seconds a vivid sense of warmth, then tingling and a sense of swelling. The radiation of pain did not correspond with the radiation of heat and tingling.

If sensory nerve trunks were a common source of pain, we should expect tumours invading nervous tissues to be painful. But in the following case reported by Rose (1954) a schwannoma which disorganized the sciatic nerve was painless.

CASE I.—A woman, aged 37 years, had a firm, rounded, painless swelling on the back of the left thigh for six months. Pressure on it caused tingling in the outer side of the foot, but there was no sensory or motor loss. At operation there was a partly solid and partly cystic tumour, which had so stretched the lateral division of the sciatic nerve that its fibres were unrecognizable. The mass, which appeared to be malignant, was excised and found to be a benign schwannoma.

Woodhall (1954) has lately reminded us of the dictum of Lewis and Hart (1930) that a tumour of a peripheral nerve usually presents as a mass which is tender but not spontaneously painful. Conway and Smith (1951) reported a fatal malignant tumour of the sciatic nerve, which was painless for the first six months. Pain has not been an outstanding symptom of 343 benign nerve tumours (Craig and Dodge, 1954) or of 31 malignant tumours (Vieta and Pack, 1951). Money (1950) observed that pain was absent in 42 out of 75 nerve tumours of all kinds. Only two out of six neurogenous tumours in the neck were painful (Conley, 1955). A tumour of the Gasserian ganglion was painless for the first two years (Cuneo and Rand, 1952). Many other authors have also reported painless tumours of sensory nerves (Kelly, 1956).

Tumours of Nerve Roots.

Pain was not conspicuous in the following case of a neurofibroma which compressed both the cord and a nerve root.

CASE II.—A woman, aged 55 years, who had been treated for rheumatism for two years, had had some fleeting pains in the shoulders, and for a time the right hip joint had been painful. For six months there had not been much pain, but her legs were getting weak and numb. An X-ray examination showed that the twelfth dorsal foramen on the right side had been eroded by a tumour (Figure I). Mr. J. B. Curtis operated and removed a huge dumb-bell tumour, which had destroyed the nerve root.

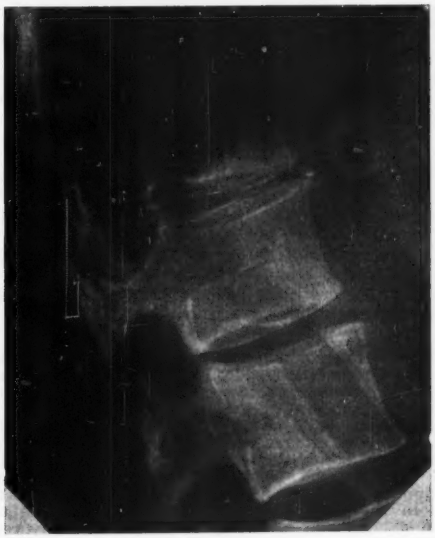

FIGURE I.

Skiagram showing enlargement of the foramen between the twelfth dorsal and first lumbar vertebræ.

A great many authors have reported painless tumours of the cord, many of which pressed on nerve roots, in every region from the foramen magnum to the cauda equina (Rogers, 1935; T. S. Kelly, 1937; Kelly, 1956). Painless paraplegia has been caused by an intradural granuloma (Bucy and Oberhill, 1950), an intradural lipoma (Taniguchi and Mufson, 1950), and an extradural cyst (Cuneo, 1955). In a ten-year history, an intraspinal dermoid cyst caused only occasional attacks of pain in the back (Ramamurthi and Anguli, 1954). Pain was late or absent in 18 out of 33 intramedullary tumours (Weersma, 1954); in six of 39 lymphomata of the epidural space (Love *et alii*, 1954); in 72 of 179 tumours of the cervical cord (Webb *et alii*, 1953); in eight out of 15 tumours of the medulla oblongata (Cooper *et alii*, 1952); in 11 out of 48 tumours of the cauda equina (Toumey *et alii*, 1950); in four out of six tumours of the foramen magnum (Smolik and Sachs, 1954).

I can be accused with some justice of raising objections which are not new. It has been known for years that

tumours pressing on nerve roots might be painless. But I believe that the significance of this has not been properly appreciated and that it has not been satisfactorily explained. When we spring to the conclusion that a pain of doubtful origin is due to pressure on nerve roots, do we keep in mind the fact that one-third of all tumours which invade nerve roots are painless? If pressure of this kind is the common pain stimulus, these tumours should be consistently painful. If pain is absent from only a few such cases, the theory must be wrong.

Five Kinds of Effective Stimuli.

In colic and nerve root pain it is permissible to assume that the diseased tissues are bringing mechanical pressure to bear on the receptors. But the pain of most diseases is spontaneous and we cannot blame external stimuli. We can only proceed by analogy and assume that pressure is operating in a manner we cannot understand. Many of us have got into the habit of regarding pain as completely specific like the other senses. Each of them can be aroused from only a single kind of receptor, and each responds to a single kind of stimulus and to no other. But pain can be aroused in undamaged tissues by five different kinds of external stimuli: (i) puncture of the skin, (ii) heat on the skin, (iii) pressure on deep tissues, (iv) touch on mucous membranes, and (v) chemical agents on mucous membranes. The pain plexus has been accepted because no other structure could have such a wide range of responses. I specify undamaged tissues because damaged tissues quickly become hyperalgesic and painful to gentle pressure and to mild thermal and chemical stimuli.

The Effect of Hyperalgesia.

When we postulate pressure as the sole mechanism, we ignore the more likely role of chemical agents. Yet in hyperalgesic tissues the pain receptors are continuously under stimulation by chemical agents (Lewis, 1942; Hardy *et alii*, 1950). In the following experiment the reactions of the epithelium and the deeper layers of the dermis immediately before they became hyperalgesic differed in quality from their later reactions.

Observation 4.—While I was polishing my car I struck a sharp projection and cleanly knocked out from my palm a circular piece of epidermis one inch in diameter. Immediately afterwards the sensation of the epidermis right up to the edge of the wound was normal to touch, pin-prick, heat or cold. The part denuded of skin had no sensation of touch, heat or cold, and it could be pricked anywhere without pain. Within ten minutes the wound had become hyperalgesic, and pin-prick to the epithelium at the edge gave an abnormal response. The dermis in the base of the wound became hyperalgesic to touch and heat, and especially to pin-prick. They remained thus until the wound had healed, six weeks later.

Much modern pain research has been done on skin which had had its epithelium scraped off, or on tissues which had been damaged in other ways (Woollard *et alii*, 1940; Bishop, 1945; Landau and Bishop, 1953). We should bear in mind that the responses recorded could not have been physiological, because the tissues must have been in a continuous state of hyperalgesia.

We have known for years that it is only in hyperalgesic tissues that pressure of ordinary magnitude is capable of inducing pain. Yet this is somehow regarded as confirming rather than denying the specific function of pressure. If the doubter asks why œdematous tissues are not painful, he might be told that the complete pain mechanism can develop only in the presence of certain chemical agents; which means, in effect, that tension is not a self-sufficient stimulus in the same way that light or sound is.

In an appreciable number of painful states when pressure cannot be convicted it is fashionable to postulate ischæmia as the sole cause. Ischæmia was believed to generate in muscle a chemical agent (factor P), which acted on pain nerve endings (Lewis, 1942). But evidence suggests that constriction pain in working muscle is due not to ischæmia, but to hyperalgesia of partially denervated muscles (Kelly, 1955). It is a remote effect of compression of a nerve trunk.

A Philosophy.

The pressure theory has never been proved; it is generally thought so self-evident that proof is superfluous and an inquiry like this is unnecessary. It is more than a theory; it is a philosophy which guides our thinking. It is held in spite of dozens of facts which contradict it. Each such fact is treated as exceptional and is explained by a supplementary theory which assists in upholding the basic philosophy. We may be told that tumours pressing on nerve roots are not especially painful because the tension has developed slowly and living tissues are adaptable. Why, then, cannot the same adaptability be called on in a case of sciatic pain which has persisted for years—because the blood flow (or the lymph flow) has probably been blocked in one and not in the other. What guides this unpredictable circulatory factor—the equally unpredictable release of chemical substances like bradykinin, histamine and acetylcholine? When forced to his last line of retreat, a protagonist may tell us that some individuals notice low-grade pain less than others.

Combatants on either side seldom realize that the theory has three unrelated aspects. Defenders often shift their ground unwittingly, and when hard pressed on the nerve-root front they might find themselves fighting effectively on the tension front. So many admittedly imponderable factors can be rallied to the cause that the final result must be confusion.

Simplification Necessary.

Nearly all progress in science consists of simplification and the establishment of principles. We can never advance in the field of pain until we simplify it and establish some principles. We should be able to discuss the question as we discuss vision, in which the principle is accepted that photic stimulation of rods and cones gives vision. We know that there is a substance known as rhodopsin and that biochemical changes take place in this and other substances. But we shall get lost if we let the main principles become confused by biochemical data or by details of the circulatory state of the retina or the composition of the body fluids.

Similarly with pain. We need fundamental principles to guide us, but we know that these principles cannot concern themselves with such minor points as the circulatory state of the tissues and transient chemical changes. We know also that there is such a quality as subthreshold pain. But its existence does not invalidate any principles I may propound about the mechanism of conscious pain, any more than the existence of a subthreshold photic stimulus invalidates the accepted principles of visual sensation.

Pain is seldom due to a single mechanism. Its causation is multiple, and several subthreshold factors often summate to give threshold stimulation. When pain is clearly caused or intensified by pressure, we often find on investigation that the painful tissues have previously been hyperalgesic.

Summary.

As responses to external stimuli, pressure sense and deep pain are related but not identical.

By analogy, the spontaneous pains of most diseases are often attributed to pressure. In different tissues, however, three unrelated mechanisms are called pressure: constriction of nerve roots; compression of terminal fibrils by smooth muscle; tension of inflammatory œdema.

Tumours of nerve trunks are often painless, as also are one-third of all tumours of the cord. Therefore, pressure cannot be the primary stimulus in root pain.

Pressure is frequently a secondary factor in tissues whose pain receptors are already sensitized by chemical agents (i.e., hyperalgesia).

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SEASONAL CONCEPTION RATES IN AUSTRALIA.

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THERE is some evidence that mammalian reproduction is modified by subtropical and tropical environments. The calving percentages of cattle in the Northern Territory vary between 40% and 70%. In the tropical sheep country of Queensland, each 100 ewes pregnant during the hot season produces 30 to 60 lambs of small size (Moule, 1954). This dwarfing has been confirmed experimentally by Yeates (1953) in Romney ewes and found to be the result of heat independently of food supply. Rats (Macfarlane, Pennycuik and Thrift, 1957) and rabbits (Shah, 1956) resorb about half their fœtuses when exposed to hot environments at 95° F. (35° C.), without prior acclimatization.

These observations suggest that there may be modifications of human reproduction in hot environments. Mills and Senior (1930) have maintained that there is a seasonal fluctuation in conception pattern in the United States, and they point out that similar changes have been recorded in Japan and Switzerland. A mean air temperature of 70° F. was considered by Mills to be the neutral zone above which fertility decreased; and also below 40° F. there was less fecundity.

Since Australia has a fairly homogeneous European population living between the latitudes of 11° S. and 44° S., it offers a unique standing experiment in human ecology. Australia is nearer to the equator than the United States of America, so that the effects of heat should be greater than those Mills recorded. Data have been collected on apparent conception rates and on birth weights in the eastern regions of Australia as indicators of fertility and of the possible effects of climate on gestation.

Methods.

Queensland official statistics of date of birth were collected, and with the assistance of the New South Wales, Victorian and Tasmanian statisticians, comparable groups from the eastern States were compiled. The date of birth was assumed to follow 272 days after conception. The years 1927-36 and 1937-46 were examined. The earlier group was chosen since contraception was little used or inadequate at that period. The 1937-46 conceptions were distributed in a similar fashion to the 1927-36 group in spite of the intervention of war. During the periods studied, there were 257,875 births in Queensland, 900,445 in New South Wales, 664,487 in Victoria and 100,106 births in Tasmania. The annual mean conception rate of each zone was used as a base-line of 1000, and the monthly deviations from this mean indicated seasonal effects.

A second examination of the possible influence of heat on the birth rate was made by comparing Queensland towns of nearly equal population and social structure in different environments. The three pre-war years 1938-1940 and the five post-war years 1948-1952 inclusive were studied.

As a third test of the effect of heat, seasonal summer-winter differences of conception in towns with high mean monthly 9 a.m. temperature in summer, Townsville and Rockhampton, were contrasted with Toowoomba, where the summer is cooler. These towns are of comparable size and social structure.

Hospital records of birth weights for the years 1948-52 inclusive yielded monthly totals of live birth weights. Premature births were included, but stillbirths were omitted in calculation of the means.

Results.

Apparent Conception Rates in Different Seasons.

The monthly means of births recorded during 20 years in the eastern States of Australia were obtained. From these the seasonal distribution of the probable conception

time of these infants was derived and plotted in Figure I. The inland regions have been separated from coastal and metropolitan zones, since the extremes of temperature are greater to the west of the dividing range than they are on the coastal strip, and a greater fluctuation about the mean temperature occurs inland than on the coast.

There was a minimum monthly conception rate in the northern region about February to March, when the greatest thermal stress is imposed during the humid heat of February and March on the coast. In the central hot-dry country the minimum occurred in December to January, during the heavy insolation at midsummer.

The difference between summer and winter conception rates on the northern coast was 14%, and for the northern inland region 22%. In southern Queensland there was a February-March minimum, but this was only 8% below the winter maximum. In New South Wales there was only a small flux about the mean, whereas in Victoria there was a detectable winter minimum. This was more marked on the coast than inland. Tasmanian conceptions showed a 20% reduction in July below the December maximum.

In all metropolitan areas there was a March-April depression of conception followed by a May peak. This may be linked with Lent and subsequent Easter marriages.

Comparison of Towns.

Another approach was made to the study of correlations between climate and reproduction in man by comparing towns on different latitudes.

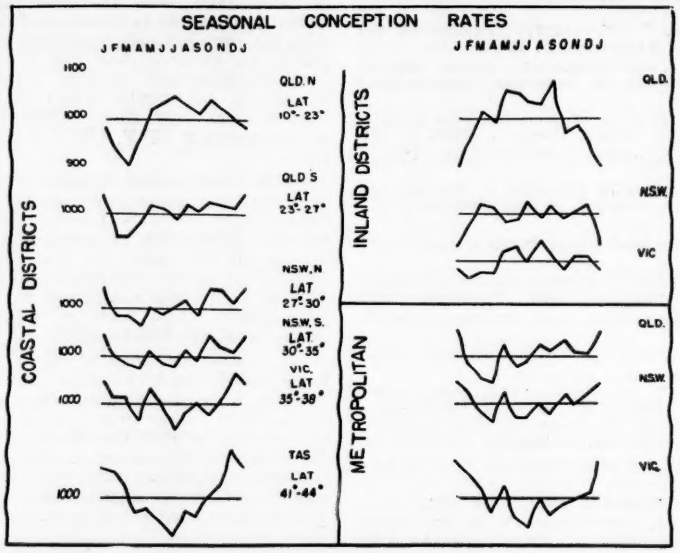

FIGURE I.

Changes in monthly conception in Australia related to the annual mean (1000). On the coastal regions the minimum conception rate in the north was in February-March and this changed to July in Tasmania. Inland districts showed a northern minimum in December-January. Inland New South Wales had a similar, though less marked, minimum at midsummer. In Victoria there was little difference between summer and winter. The large towns of the east coast showed a February-March minimum of conception, moving towards a winter minimum in Victoria.

Table I summarizes the effects of latitude. There was a highly significant reduction of yearly births in tropical Townsville and Charters Towers below those of Toowoomba and Stanthorpe, in the southern hills. The populations are nearly equal and ethnically there is much in common between the two pairs of towns. When correction was made for the immigrant school population of Charters Towers, there was still a significant reduction in annual births below those of Stanthorpe.

TABLE I.

The Relation of Birth Rates to the Thermal Environment and Latitude of Pairs of Towns of Nearly Equal Population in the North and South of Queensland.

Town.[1]	Latitude. (° S.)	Mean Summer Temperature. (° F.) D.J.F.[2]	Relative Humidity. (Percentage.) D.J.F.	Mean Winter Temperature. (° F.) J.J.A.[3]	Relative Humidity. (Percentage.) J.J.A.	Mean Annual Population. (8 yr.)	Total Births (8 yr.)	Birth Rate per 1000 of Population.	P.
Townsville	19	81·3	73·3	69·2	71·0	34,280	5723	20·8	0·001
Toowoomba	27·5	71·1	72·8	51·9	74·8	34,250	6186	22·5	
Charters Towers	20	81·7	65·5	65·2	62·8	7800	1071	17·1[4]	0·001
Stanthorpe	28·5	69·5	61·0	47·0	71·7	7560	1571	25·9	
Innisfail	17·5	79·5	79·0	66·7	85·7	13,300	2527	23·8	0·050
Longreach and Cloncurry	21	88·1	46·0	63·5	48·0	12,700	2574	25·3	

[1] Toowoomba is at an altitude of 1920 feet and Stanthorpe 2600 feet. The other towns are near sea level.

[2] D.J.F. = December, January, February.

[3] J.J.A. = June, July, August.

[4] Subtracting a secondary school transient population of approximately 1500 from the population of Charters Towers, the birth rate per 1000 population becomes 21·1, which is significantly different from that of Stanthorpe (P = 0·001).

Comparison of Innisfail (wet, hot) with the combined populations of Cloncurry and Longreach (dry, hot) indicates a slightly lower birth rate on the coast than inland. All three towns are within the tropics. The average humidity is nearly twice as high on the coast as at Cloncurry.

A difference between summer and winter birth rates occurred in towns exposed to high summer temperatures (Townsville, Rockhampton), but was not found in Toowoomba where the height above sea level reduces the summer temperature (Table II). The difference between summer and winter conception rates in Townsville and Rockhampton was 7·7% and 8·1% respectively, and these differences are significant at the 5% level.

Birth Weights.

On analogy with sheep and cattle, the offspring of which are reduced in size by gestation during summer in the tropics, a survey of human birth weights in Queensland was undertaken (Table III). The variation is great, and standard deviations are at least ± 1 lb. 2 oz. about the mean. There is a small, statistically insignificant, difference between birth weights in the north and south.

Discussion.

The seasonal pattern of conception rates with subtropical maxima in winter and cold temperate maxima in summer described in the United States (Mills and Senior, 1930) appears also in Australia. There is no known single cause.

It is possible in the male, that high testis temperatures or body temperatures reduce fertility or frequency of mating. It is difficult to estimate such effects, though it is known that sperm production is reduced in man during and after hyperthermia (MacLeod and Hotchkiss, 1941). The scrotum of man in a hot environment sweats and the surface temperature probably remains below blood temperature. Few men work so hard in the heat that the blood temperature is significantly raised, so that male infertility seems unlikely to be the full answer. In Tasmania the minimum conception rate (occurring in winter) would have to be ascribed to inactivation of spermatogenesis or of sperm by cold, though there is no evidence for this in the temperature range of Tasmania.

In the female there is the possibility of reduced ovulation in the heat. This has been observed in the acclimatized rat (Macfarlane et alii, 1957). Resorption or abortion have been observed in heated rats, rabbits and ruminants, and they could possibly be induced in man by summer temperatures. Such data are difficult to collect in human societies, however. Presumably the seasonal acclimatization is insufficient in some members of the population to protect the fœtus against endocrine disturbance induced by heat or cold.

Religious customs could account for some of the Brisbane February-March minimum (Lent), but not for the inland December-January minimum of conception. It is more likely that the heat load is responsible for both periods of reduced apparent conception—humid heat lasting till March on the coast and dry heat reaching its maximum during December in the central regions.

It appears that man achieves optimal fecundity in a temperate climate and higher or lower temperatures seasonally reduce the reproductive yield. Comparison between towns of equal population in Queensland indicates that winter-summer differences are eliminated in places like Toowoomba where altitude reduces summer temperatures. Summer-winter differences of conception occur in tropical towns where summer heat is above comfort level for four to six months. There are, also, significant differences between the low birth rates in tropical towns and the higher rates of subtropical towns in the hills. In the Townsville-Toowoomba comparison, the birth rate would be lowered by the number of retired people beyond the reproductive age living in Toowoomba, yet this southern town has the higher birth rate. Climate appears to have a significant effect in this case, as it does in the comparison of Charters Towers with Stanthorpe.

It is difficult to compare the industrial with the non-industrial towns, or cattle country with cane field areas, since the population, age, sex and social components differ widely. In the towns chosen for comparison, however, the degree of homogenity is sufficient to suggest that the thermal environment has been the main determinant of the reduced tropical urban birth rates. The rural tropical birth rate is, over-all, higher than that of urban subtropical Queensland. This higher rate is the product of a rural or small town fecundity that occurs in all countries, whereas the large-town effect (Brisbane) is to lower birth rates, whatever the environment.

Although lambs and calves resulting from summer gestation are smaller than those produced from winter pregnancy, there was no clear human response of this type in Australia. In South Africa, Salber and Bradshaw (1952) could detect no consistent seasonal change of birth weight amongst European, Bantu or Indian infants. The human mother is not exposed to the solar radiation that adds considerably to the heat load of ewes and cows living in the open during summer, and she probably avoids the extremes which could alter birth weight.

Summary.

1. Conceptions were reduced during summer (December-January) 22% in the inland districts of north Queensland relative to the maximum winter rate of conception. In northern coastal towns the summer minimum of conception occurred in February-March.

2. In Tasmania conception was high in summer and low in winter, while intermediate coastal zones between Tasmania and north Queensland show intermediate fluctuations of conception with season.

3. In Queensland there was a significantly higher birth rate in hill towns of the southern subtropics than in similar towns to the north in the coastal tropics.

4. In tropical towns there was a significant reduction in summer conception rates relative to winter rates in the same towns.

5. There was no major reduction in birth weights in the tropics.

TABLE II.

The Summer and Winter Conception Rates in Townsville, Rockhampton and Toowoomba.

Town.	Latitude. (° S.)	Mean Temperature. (° F.)[1] D.J.F.[2]	Relative Humidity. (Percentage.) D.J.F.	Mean Temperature. (° F.) J.J.A.[2]	Relative Humidity. (Percentage.) J.J.A.	Mean Annual Population.	Conceptions (8 yr.). Summer. D.J.F.	Conceptions (8 yr.). Winter. J.J.A.	P.
Townsville	19	81·3	73·3	69·2	71·0	34,280	1438	1548	0·05
Rockhampton	23	80·7	67·2	63·5	70·5	36,950	1488	1608	0·05
Toowoomba	27	71·1	72·8	51·9	74·8	34,250	1554	1527	Not significant.

[1] 9 a.m. monthly mean temperatures in Stephenson screen.

[2] D.J.F. = December, January, February.

J.J.A. = June, July August.

6. It is probable that heat in some fashion was responsible for the reduction of conceptions during summer in tropical Queensland.

TABLE III.

The Average Birth Weights of Infants Born During Five Years in Cairns and Brisbane.

Town.	Latitude. (° S.)	Number.	Average Birth Weight.
Cairns	17	3068	7 lb. 7·6 oz. (3·390 kg.)
Brisbane	27·5	45,400	7 lb. 12 oz. (3·515 kg.)

Acknowledgements.

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CHILD GUIDANCE AND THE PSYCHOLOGICAL LABORATORY.[1]

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THE purpose of this communication is to illustrate the use of a psychological laboratory in dealing with simple behaviour problems of children. The term "psychological laboratory" is used to cover the space, equipment and personnel used in exploring psychological problems. It consists of a room equipped with apparatus for testing, which includes, in addition to the standard material of intelligence tests and projection tests, sandpits, toys, puppets, drawing materials, etc. The personnel consists of a psychologist who understands the special disciplines involved, and a social worker to investigate home conditions when necessary.

The questions we will ask ourselves are: Do the behaviour problems of children warrant the provision of the facilities described above? Are they of material assistance in diagnosis and treatment, and do the benefits justify the expenditure involved?

In order to illustrate the clinical material, seven cases are described as representative types seen in practice.

[1] Delivered at a meeting of the Clinical Society of the Brisbane Children's Hospital on February 17, 1959.

CASE I.—*John, aged 11 years, was brought on account* of incorrigible behaviour at home. He was aggressive and *fought with his brothers more than "seemed natural".* He was enuretic and a restless sleeper. *His school work* was below standard. On examination, interrogation was *difficult. He seemed disinterested and would not cooperate.* Questions were stone-walled. Emotion and thought seemed at variance. As might be expected, his parents were *extremely worried.*

In the laboratory, Wechsler-Bellevue and thematic apper*ception tests revealed conceptions of interpersonal relation*ships as either hostile or exploiting. Phantasy was destructive and anti-social. Feelings of rejection and *impulsive reactions towards violence prevailed. The* thinking was schizoid, and social relationships were characterized by hostile withdrawal. The psychological findings were in keeping with the clinical impressions. The boy was obviously out of touch with reality, and was *diagnosed as a case of early schizophrenia.*

Treatment in the form of encouragement, and discussion *with parents towards the alteration of environmental* difficulties, were unproductive. There was a disparity in the parental viewpoints which could not be lessened. *Neither would admit faults, though both were interviewed* separately and together. It was all too obvious that they were not in tune with their son, and little change could *be expected without drastic reorganization of the family* set-up.

After the avenues for a complete change of environment were explored, it was suggested that the boy be sent to relatives in the country. By good fortune an aunt had a farm, and her grown-up *son promised to collaborate in* making the child both welcome and useful.

He has now been a year away from his parents, who admit that there is a marked improvement. He is less inhibited in speech and converses quite freely about farm life. Emotion and thinking are patterned on more normal lines. There is hope that the steady advance of schizoid *trends may have been halted.*

CASE II.—Dora, aged eight years, came to us on account *of frequent nightmares.* Her home life was very unhappy; the father was a drunkard, who often attacked his wife and threatened to burn down the house. The mother was herself under psychiatric care, but on account of her responsibilities toward the children, could not enter *hospital for treatment.*

The psychiatric interview revealed considerable insecurity and anxiety. Dora complained of tigers chasing her in her dreams; she said she was not afraid of her father, although it gave her a headache to listen to her mother and father fighting.

In the psychological laboratory, a play session revealed that Dora was deeply attached to her father, and was very insecure both as to her immediate safety and as to her place in her mother's affection. Her intelligence shown by the Binet test (Stanford revision) was very superior.

The social worker visited the home, and throughout the ensuing two years kept in touch, thereby helping the mother to maintain a higher morale than would otherwise have been possible. Her subsequent divorce and remarriage posed fresh problems for Dora. She was brought to the clinic again by her mother on account of bouts of weeping and generally difficult behaviour.

It was discovered by the psychologist that she resented her new father, and grieved over her real father. She also believed her mother no longer loved her.

The social worker, after visiting the home, brought the information that the mother was very happily remarried, but that in her joy lay seeds of truth in Dora's remarks—she had no eyes save for the new husband.

Eight years after Dora's first visit, the social worker made another call at the home as a part of our periodic surveys. It was at a crucial time. She was about to sit for a public examination, and was distraught with anxiety because her mother had told her she had to leave school at the end of the year. Dora's ambition was to become a

teacher after a further period of two years at school. At this juncture it was difficult to see how Dora could encompass her ambition without straining the family exchequer. An underlying emotional difficulty was the mother's resentment at the need for her present husband to provide for Dora's future.

Dora and her mother were seen several times. The situation was explained, and we obtained for her a position in a teaching institution. Dora's symptoms subsided. Both parents then agreed to permit the child to continue at a high school.

CASE III.—Jimmy, aged four years, was brought for treatment by his aunt, on account of lack of bowel control. He frequently passed fæces into his pants. Parental control had been inadequate. His irresponsible mother was addicted to bouts of alcoholism, and as a result he gravitated to the custody of an aunt and her husband.

After the psychiatric interview, treatment by planned routine of visits to the toilet was suggested, but in spite of encouragement to both patient and his relative, there was no improvement.

It was decided to make further investigation in the psychological laboratory. This was instructive. Given his choice of the numerous toys in our equipment, he chose the filling of trucks with sand and then "shooting off" the surplus. The man was placed on a gun; the boy belched and said "a gun does pooh like horses". On the following visit he played with tigers who evacuated; he sat boys on toy commodes and discoursed on cow pooh and people's pooh. The shooting and truck incidents were repeated. On the next occasion the pooh games were less prominent. Play was more normal.

During the period of the tests, the aunt was advised to discontinue bowel training and to make no comment other than referring to a "chamber" available in the garage. She was asked to express the utmost affection and tolerance of the child even when he soiled his pants. The boy was to be accepted *in toto*.

Within six weeks the boy was completely clean, and used the chamber as a matter of course.

There seems to be no doubt that the improvement in the boy's condition was due to his being allowed to play out his fantasies without fear of hindrance, and to the improved environmental condition which resulted in replacing tension and insecurity with kindly understanding.

CASE IV.—Dorothy, aged eight years, was a very unhappy child. Her mother said she would not mix with other children, and since birth had wet the bed every night.

In the consulting room, Dorothy was evasive. She answered questions monosyllabically, and could not be induced to give a coherent account of likes and dislikes. She was facile, emotionally dull and generally unrevealing. Her unhappiness was definite, but the causes could not be brought to the surface.

At the play interview in the laboratory, Dorothy revealed that her cousin spread the news of her bedwetting at school, and the children pointed to her in scorn.

She slept in the same room as her younger sister, and had to strip her bed when it was wet: "This was awful."

The mother was advised to give the child her own room and encourage her to believe that it was entirely hers. Easier standards of conduct were suggested, and the parents were asked to demonstrate more affection for the child.

In three weeks the enuresis had disappeared; the patient was happier and made friends with her school mates.

There was an interesting sequel in that a year later enuresis returned, but promptly disappeared on the exhibition of amphetamine. There were no events of emotional significance to account for her lapse, and apparently a simple course of sleep lightening achieved relief.

CASE V.—William, aged 10 years, was referred for periodic attacks of asthma, which had become increasingly frequent and interfered with his school work. Examinations were a cause of worry, as owing to illness, he seldom completed them.

Examination in the consulting room revealed a poor physique, but no physical signs indicating asthma at the time of examination. The boy admitted that he had frequent asthmatic attacks, but could not associate them with any particular emotional state.

The father appeared to be both kindly and thoughtful. From his description the mother was preoccupied with the child and somewhat ineffective in her management. Previous medical advisers had given antispasmodics, which though helpful, had not prevented the frequent occurrence of crippling asthmatic attacks.

In the laboratory a play interview revealed that asthma occupied the foreground of the child's attention. He actually enjoyed the convalescent period when he was relieved of chores and was waited on by his mother. The mother was revealed as frustratingly overprotective and apprehensive. She literally kept the boy in a state of insecurity by suggesting that he would get asthma.

As a result of the clear indication that the maternal attitude was at fault, the father was advised to send the child to a relative in the country. The attacks promptly disappeared, and on medical advice the child's stay has been lengthened.

The respite will give opportunity for the re-education of the mother, which seems the essential preliminary towards better adjustments. It is commonplace that after a period of stress a holiday enables us to reshuffle our thoughts, and we can return to our environment in a hopeful frame of mind. The absence of the child serves a similar purpose by breaking vicious circles in interpersonal relationships.

The major factors for future decisions relate to the length of stay away from home and the details of parents' re-education.

CASE VI.—Tommy, aged four years, was brought to us on account of stuttering, which had begun twelve months previously. His speech commenced at the age of 12 months, and was natural and fluent by the time he was three years old. The clinical impression at the first interview was one of normality. It showed a bright boy, interested in his surroundings, with surprisingly little speech defect beyond a hesitancy which could be overcome.

The history, as elicited from the mother, revealed that Tommy's younger brother Ken had "jammed his finger" in the gate on to the road twelve months earlier. Shortly afterwards, he escaped into the street. Tommy followed him, in an endeavour to bring him back out of danger. The mother saw the two boys up the road, and gave chase. She punished Tommy severely for leading his brother into dangerous traffic. It was not until later that she learnt the true version of the incident. But Tommy's stutter began from this episode.

It seemed improbable that a stutter of this description could be produced by one incident, but in the psychological laboratory it was possible to produce the stutter by getting the child to cooperate in playing with cars and trucks. After a brief spell away from this, play with chalk and blackboard (not drawing motor cars) produced smooth, unobstructed speech.

The experiment was repeated three times, with similar results. Further interrogation revealed that the family house was on a highway, which carried a considerable volume of traffic. The mother's efforts at instilling fear of traffic had been assisted by her husband, who was also accident conscious.

The combined clinical and laboratory evidence pinpointed an emotional situation of a restricted but quite definite type.

The parents were cooperative. They agreed to "play down" the traffic danger angle, and as a further step, the father is considering living in a district with fewer traffic hazards.

CASE VII.—Joe, aged four years, was referred on account of his feeding habits. He ate his food with extraordinary slowness. Each meal was a battle between mother and child, which lasted for an hour or more. Mealtimes became dreaded pitched fights between Joe and the rest of the family. He would eat only when his mother obeyed his plea: "You feed me."

As a baby and a toddler he had a good appetite, but there had been feeding incidents. For the first three and a half weeks after the mother brought him home from hospital he vomited all food. It was then discovered he had a pyloric spasm. After treatment feeding proceeded successfully. Weaning was uneventful; he advanced from "bottle to cup" by 12 months.

Joe was two years old when his sister was born. Until this time his mother had spoon-fed him. Upon her return with the baby, he was suddenly expected to feed himself. From this time onwards serious feeding trouble began. The child had discovered that feeding presented an opportunity to keep his mother close to him, and at the same time have the "edge" on his younger sister.

The mother proved to be extremely cooperative and carried out our instructions. She was advised to give him lunch out of doors in a play tent with his sister, and to make it part of a game both children were playing. She herself was to keep inside the house. In addition the mother was also told to cease her admonitions about eating, and to ignore his full plate at the end of a meal when the family dined together. Furthermore, it was suggested that she try to make him feel she loved him even more than his small sister. He was to be encouraged to grow up, as it was fun to be no longer a baby.

After three weeks of rigid adherence to this treatment, he was reported to be "now no trouble at all" and "getting fat!".

Comment.

All the foregoing cases, after clinical examination by the psychiatrist, were referred to the psychological laboratory. Although this was routine in a department devoted to research, the procedure was advantageous in therapy in each case.

It appears to us important to note that, excluding routine checks, no patient was seen more than five times; this indicates that a laboratory expedites treatment. Economy in time comes from the advantage of a planned attack involving child testing under conditions favourable to parent and child cooperation. The result is that the parents are more rapidly convinced of their own mismanagement and are readier to accept advice.

It may be noted that major environmental alterations were made in three cases, two children being sent to the country, and in the other case, parental change of domicile being advised.

The coincidence of clinical impressions and psychological tests create a certainty of judgement, which permits the giving of forthright advice without quibble or delay. When major environmental changes are involved, one is tempted to procrastinate in the hope that simpler advice will be of benefit. Often shortage of time due to an overfull case load forces us to tread the easy path of procrastination, but as morbid interpersonal relationships are cumulative, such a course is dangerous.

Unlike many disorders in medicine, with behaviour disorders time is not usually on the side of the patient. Time often leads to the brink of disaster, rather than to the healing of psychological wounds. Time must also be considered from another angle. The increase of new knowledge, whilst clarifying many old problems, gives psychiatrists more responsibilities. New problems, new hazards, new concepts and new therapies (if we except the electroconvulsive therapy machine) make inroads on his working hours. The success of modern psychiatric therapy has made greater encroachment since more patients ask for treatment, and this increases the case load. There is therefore a definite need to search for methods to shorten the time required for efficient diagnosis and treatment. One of them is to install the psychological laboratory.

One of us is old enough to remember the days when the psychiatrist did his own testing, both pathological and psychological. It was a pleasant relaxation when tests were few. Today such a course spells frustration, and would reduce effective work loads to a trickle. The psychiatrist must encourage other specialists to work in the field, though retaining a coordinating position for the new knowledge which they acquire.

Let us review the cases with this in view.

Case I.—The decision as to the type of treatment was undoubtedly helped by the psychological tests, which revealed schizoid trends and grave difficulties in interpersonal relationships. Schizophrenia in childhood is a serious condition, which necessitates prompt and firm handling. Laboratory findings undoubtedly assisted certainty in diagnosis.

Case II.—The case of Dora illustrates the need for a follow-up investigation by the social worker. Interpersonal relationships are never static. The relief of symptoms may be temporary, and relapse may occur under strain. Not the least of the virtues of the old-time family doctor was his accessability and knowledge of the family unit throughout many years. There is a great need for the psychiatrist to keep in touch with patients prone to relapse. It can be achieved only by having a social worker in the psychological laboratory team.

Case III.—Attempts at the treatment of Jimmy on conventional lines failed. It was the freedom to achieve his urges in the psychological laboratory with its playroom atmosphere which set him on the road to recovery. The child did not reveal his phantasies during conversation in the consulting room, yet a knowledge of their details was as essential to therapy as would be the type of bacillus in a medical case. The need for laboratory procedures in both cases seems obvious.

Case IV.—During the preliminary psychiatric interviews, Dorothy talked on the "yes", "no" level, which is unrewarding. The clue concerning the devastating influence of her cousin's scandal and the sleeping habits of the patient were revealed not in the consulting room, but in the laboratory. It was this information which enabled us to make satisfactory adjustments in the environment.

Case V.—During the initial psychiatric examination of William, a presumptive insecurity and a submerging mother were revealed. The child's testing in the psychological laboratory confirmed and enlarged on this; he was obsessed by thoughts of asthma and his next attack. His life revolved around this morbid theme. As a result of the combined approach, the need for a complete change of environment seemed to us obvious. It was not surprising that the previous therapy by drugs had failed. The interlude of separation not merely enables the child to cut his own vicious circles, but gives a possibility of parent re-education, which may allow him to remain asthma-free when he returns.

Case VI.—The story of Tommy admirably illustrates the precision which can be reached through the use of suitable tests. Just as in clinical pathology reactions can be produced in the test tube, so is it possible to repeat behaviour responses in the psychological laboratory. As a result, treatment can be advised on a rational, not an empirical, plane.

Case VII.—Joe's history illustrates the use of the laboratory as a time-saver for the psychiatrist. It was the careful hour-to-hour survey of the child's feeding habits which led to the exact determination of the mechanism involved, and to the parent's realization of her errors. The rapid manner in which the symptom was cured was due entirely to the careful case taking and the *rapport* which was achieved. As a result, the parent rapidly cured the child.

The foregoing sample of cases confirms the thesis that the psychological laboratory has an integral place in child

guidance. It leads to more efficient treatment, expedites recovery and warrants the expenditure it entails. The psychological laboratory is, indeed, an essential in dealing with behaviour problems of children as is a pathological laboratory in delving into their physical complaints.

STAPHYLOCOCCUS AUREUS FROM AIR-BORNE FIBRES IN A HOSPITAL WARD.

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THE development of a sampling device by Pressley (1958) and the opportunity to use it in a surgical ward of this hospital permitted the collection of samples of air-borne fibres and their direct examination.

The demonstration of *Staphylococcus aureus* in collected air-borne fibres would be of great interest in interpreting the role ascribed to the woollen blanket in hospital-acquired staphylococcal infection. Work carried out by Pressley had indicated that the air-borne fibre collected by his sampler was essentially cellulose (cotton) in nature. A bacteriological investigation in which viable organism counts were obtained, and a search made for coagulase-positive *Staph. aureus*, would contribute to our understanding of the staphylococcal problem.

Duguid and Wallace (1948), in their study of air infection with dust liberated from clothing, demonstrated by a slit sampler technique that air-borne organism counts rose enormously when subjects exercised in a small closed cubicle. They discussed the role of clothing in this liberation of dust-borne bacteria. Hare and Thomas (1956) extended this work, confirmed the findings and suggested that the *Staph. aureus* was derived indirectly from the nasal passages, which first contaminated the skin and clothing.

The work here reported has taken the matter a step further, with the direct demonstration of the presence of coagulase-positive antibiotic-resistant *Staph. aureus* in air-borne fibres collected at bed height in a hospital ward.

METHODS.

The sampling device was that of Pressley. This consisted of a "Ventaxia" fan, 6 in. in diameter, mounted so that it collected a sample of air-borne fibres at 34 in. above floor level—that is, at bed height. Air was drawn by the fan through a "Terylene" filter stretched tightly across the fan inlet, and the air-borne fibres from the vicinity of the sampling device were collected on the filter.

The samples were collected in a surgical ward with the sampler two feet away from one of the six beds in the ward, just inside the doorway. Times of sampling are shown in Table I. Bed making and ward cleaning usually began about 7.30 a.m., and the work was completed by 9.30 a.m. A large cylinder-type vacuum cleaner was used for cleaning. No oiling of floors was practised. The changing of blankets was irregular and at the discretion of the ward sister. Usually they were changed on a patient's discharge from hospital. The blankets were laundered by being washed with soap and water at 37° C. Light movable screens made of cotton material were erected around the bed when dressings were carried out.

Sterilization of "Terylene" Filter and Sampling of Collected Fibre.

In Tests A to E, the "Terylene" filters used either were new, or were washed with soap flakes and water and dried overnight in an incubator at 37° C. No sterilization was attempted. In later tests the filters were autoclaved at a pressure of 15 lb. for 15 minutes, and dried overnight at 37° C. No deterioration of filters was observed.

Non-sterile filters used in the first experiments did not contribute organisms to the fibres collected upon them. This was shown by an experiment in which cotton fibres, dry-heat sterilized (160° C. for 90 minutes), were collected on sterile and non-sterile filters respectively. Counts determined on the cotton-fibre samples from both sterile and non-sterile filters by the method set out below showed in both cases only one colony growing in sixty-four 0·02 ml. drops tested in replicate fibre samples. We concluded that fibres did not pick up any organisms from the non-sterile filters.

When sterile filters were used, the housing of the fan and the clip were sterilized with alcohol (70%).

A weighed filter was placed on the sampling device in the ward, and the sampler was allowed to run for the specified time. At the end of the sampling period, numerous fibres could be seen on the filter. The sampler was switched off and the filter removed, care being taken to avoid loss of fibres. In the laboratory the filter plus fibre was weighed.

The filter was placed in a sterile Petri dish, and by scraping with two sterile spatulas, the fibre was collected and rolled into several small balls. Two separate aliquots were transferred aseptically to two sterile tared quarter-ounce Bijou bottles and weighed. A further aliquot was taken for fibre composition determination.

Viable Organism Counts on Fibres.

Organism counts were made by the Miles and Misra (1938) method as follows.

The appropriate volume of 0·85% saline was added to the weighed sample of collected fibre to prepare a 1:1000 suspension—that is, 1 mg. of fibre per millilitre. Twofold saline dilutions of this suspension were prepared, each dilution being vigorously shaken by hand for one minute before proceeding. Because of the difficulty of keeping fibres uniformly in suspension during pipetting, dilutions were made by transferring all of each dilution to an equal volume of saline. The dilutions normally used for counts were 1:1000, 1:2000, 1:4000 and 1:8000. Counts were made on oven-dried heart infusion agar ("DIFCO") and heart infusion agar containing 7% sodium chloride. Four 0·02 ml. drops of suspension were plated on each of the two media. To minimize settling of the fibres in the pipette, it was refilled after delivery of two drops of suspension. Counts on one sample were completed before the series of saline dilutions was prepared from the replicate sample. Plates were incubated for 24 hours at 37° C., and colony counts were made with the aid of a Zeiss binocular stereomicroscope at a magnification of 10.

Coagulase-Positive Staphylococci.

Approximately 100 colonies were picked from the Miles and Misra drops of 7% sodium-chloride heart-infusion agar and plated to heart infusion agar. To prevent bias in selection of organisms which might be thought to be *Staph. aureus*, every colony in a drop was picked off until a total of approximately 100 were subcultured. After 16 hours' incubation at 37° C., colonies were tested for coagulase production by the slide method of Cadness-Graves *et alii* (1943), and smears were prepared at the same time for Gram staining.

Antiobiotic Sensitivity of Coagulase-Positive Staphylococci.—The method of testing was that of Tolhurst, Buckle and Williams (1955).

Growth was tested on heart infusion agar containing antibiotics in the following concentrations: penicillin, 1 unit per ml.; streptomycin, 10 μg. per millilitre; chloramphenicol, 10 μg. per ml.; "Terramycin", 5 μg. per millilitre; "Achromycin", 2·5 μg. per millilitre; erythromycin, 1 μg. per millilitre. A control medium consisting of heart-infusion agar was inoculated at the same time. The inoculum was prepared by diluting a 3 mm. loopful of a 24-hour heart-infusion broth ("DIFCO") culture with 5 ml. of saline, and a 2 mm. loopful of this dilution was streaked to the test and control agar plates. For the penicillin sensitivity test, an undiluted 24-hour broth culture was used. If growth was visible on the agar plate after 24 hours' incubation at 37° C., the organism was regarded as resistant to that antibiotic.

Composition of Fibre Sample.

The composition of the fibre sample was determined by the triple-dye staining method of Pressley (1958). These counts were made for us by T. A. Pressley on fibre aliquots.

TABLE I.

Sample.[1]	Total Weight of Sample. (Milligrammes.)	Time of Sampling.	Weight of Fibre Tested. (Milligrammes.)	Organism Count per Milligramme of Fibre.		Number of Coagulase-Positive Staphylococci.	Number of Organisms Tested.	Percentage Composition of Fibre.		
				Heart Infusion Agar.	7% Sodium Chloride Agar.			Protein.	Cellulose.	Other.
A 1	20·6	6.45 to 11.45 a.m.	1·2	3400	650	3	8	6·0	94·0	0
2			2·4	4900	2000					
B 1	22·7	7.10 to 9.45 a.m.	5·7	12,090	3160	15	70	1·0	99·0	0
2			6·4	14,630	7140	5	96			
C 1	22·1	2.55 p.m. to 6.45 a.m.	3·9	2270	650	0	94	2·7	97·3	0
2			4·0	1460	675	0	81			
D 1	9·0	6.45 to 11.45 a.m.	2·0	3880	2130	0	84	—	—	—
E 1	22·9	11.45 to 6.20 a.m.	2·6	11,160	6170	52	103	3·5	96·5	0
2			2·5	9350	4780	2	103			
F 1	4·5	6.15 to 10.15 a.m.	1·5	6120	2470	10	109	5·5	94·5	0
G 1	14·3	6.20 a.m. to 2.30 p.m.	2·1	6240	3320	1	113	3·5	95·8	0·7
2			1·8	4800	1430	2	65			
H 1	26·3	2.25 p.m. on August 10 to 2.40 p.m. on August 11.	3·6	17,330	11,790	1	127	7·1	92·9	0
2			3·2	7430	4950	4	118			

[1] The figures 1 and 2 represent replicate tests on the one sample.

RESULTS.

The results are shown in Table I.

Samples A to H were taken over a period of 27 days, from July 27 to August 11, 1958. Samples A to D (inclusive) were collected in bay 4. This bay contained six beds, of which an average of four were occupied during the experimental period. Samples E to H were collected in bay 6, which contained six beds all occupied, two by walking patients.

Viable organism counts were obtained by taking the average of all countable drops containing more than 15 colonies. Insufficient fibre was collected in samples D and F to permit of replicate testing.

Fibre Composition.

All samples were shown to consist predominantly of cellulose. The greatest protein (wool) count was 7·1%, the lowest 1·0%. The cellulose (cotton) content of the collected fibres ranged from 92·9% to 99·0%. It is apparent, therefore, that the air-borne fibre sampled at bed height contains an overwhelming proportion of cellulose fibre, and the role of the woollen blanket as a source of air-borne fibre or fluff must therefore be reexamined. Numerous sources of cellulose abound in hospital wards—the sheets and bed linen, the clothing of patients, staff and visitors, dressings, cellulose tissues, and ward furnishings such as curtains and movable screens. From these sources are derived the cellulose fibres which float in the air.

Viable Organism Counts.

The counts are listed in Table I as organisms per milligramme of the collected air-borne fibre. The times of collection ranged from 2 hours 35 minutes to 24 hours 15 minutes, but the differences in the organism count could not be related to the length of the sampling period. Nor could they be related to the time of the day over which the sample was collected.

The mean viable organism count on heart infusion nutrient agar found in 14 samples from eight tests was 7500 per milligramme of fibre, with a range of 1460 to 17,330, while the mean organism count on 7% sodium-chloride heart-infusion agar (that is, the staphylococcus-micrococcus count) was 3670 per milligramme with a range of 650 to 11,790. It will be noted that agreement between the results of replicate examinations on any one fibre sample was only fair; this may be due to the possibility that separate fibres carry different numbers of organisms.

Coagulase-Positive Staph. Aureus.

The slide coagulase test was carried out on 1171 organisms picked from 7% sodium-chloride heart-infusion agar plates, and 95 coagulase-positive organisms were found. Only those organisms which caused the complete and rapid (5 seconds) coagulation of fresh human plasma were recorded as "positive". Numerous agglutination reactions of suspensions were observed, but these occurred at longer time intervals, and were resuspended on mixing with the loop. It will be noted that sample E1 yielded 52 coagulase-positive organisms out of 103 tested, while the replicate sample yielded only two. This reflects the non-homogeneity of the fibre sample from the viewpoint of organism distribution, and this point is further discussed later in this paper. Coagulase-positive staphylococci were isolated from six of the eight separate samples and from each of the replicate examinations.

Antibiotic Sensitivities of Coagulase-Positive Staph. Aureus.

A total of 95 coagulase-positive colonies of *Staph. aureus* was isolated from 14 tests on eight separate samples of fibre; 94 of these were tested for antibiotic sensitivity, and the results are shown in Table II.

TABLE II.

Antibiotic Sensitivities of 94 Coagulase-Positive Staphylococci.

Antibiotic.	Concentration.	Resistant Organisms.	Sensitive Organisms.
Penicillin	1 unit per millilitre.	80·8%	19·2%
Streptomycin ..	10 µg. per millilitre.	71·2%	28·8%
"Terramycin" ..	5 µg. per millilitre.	70·2%	29·8%
"Achromycin" ..	2·5 µg. per millilitre.	70·2%	29·8%
Chloramphenicol ..	10 µg. per millilitre.	3·2%	96·8%
Erythromycin ..	1 µg. per millilitre.	0	100%

The high proportion of organisms resistant to the commonly used antibiotics—penicillin, streptomycin, "Terramycin" and "Achromycin"—is noteworthy, as is the lack of resistance to chloramphenicol and erythromycin.

DISCUSSION.

The finding that the fibre composition of air-borne fluff sampled at bed height in a surgical ward is essentially cellulose (cotton) is of great importance. Many investigations have shown that organism counts in the air of hospital wards rise considerably when beds are made and floors are swept. In a recent paper, Schwabacher and Salsbury (1958) demonstrated that the air count, as measured by plates exposed for four-hour periods in the wards, was many times greater when woollen blankets

were used than when cellular cotton blankets were used. The meshwork in the cotton blanket structure would allow cotton fibres to penetrate and be trapped in the spaces of the network. The straight, close weave of the woollen blanket, on the other hand, would not allow cotton fibres falling on to it to penetrate the surface, and thus be held. When the blanket was shaken during bed-making, these fibres would be liberated. Oiling, of course, would prevent this with woollen blankets. Knowledge of the height above ground level of the shelf on which the plates were exposed in the Watford investigations would be useful in interpreting the observed results. Pressley showed that floor dust contained up to 40% of wool fibre, but that air-borne fluff at bed height contained only 1% to 6% of wool fibre; hence the composition of the fibre falling on to exposed plates would be different according to the height above floor level at which they were exposed.

During dressing, wounds are exposed to contamination by air-borne fibres at bed height. Our investigations have shown that these fibres are cellulose, and that they carry high numbers of viable organisms, including coagulase-positive, antibiotic-resistant *Staph. aureus*. One cannot stress the quantitative significance of the numbers of coagulase-positive *Staph. aureus* organisms found, because one or two fibres containing large numbers of these organisms could be taken in one sample and be missing from its replicate. Williams (1949), who studied the hæmolytic streptococcus content of floor dust, likewise found difficulty in obtaining agreement in replicate samples because of the non-homogeneity of the material. This type of result has occurred in our series. However, the important point is that fibres carrying these organisms have been collected from the ward air, and they must therefore be regarded as potentially dangerous.

Measures adopted to stop fibre shedding from cotton goods must therefore assume a high priority in the reduction of the bacterial load to which the debilitated hospital patient is exposed. Possible methods include treatment with polyvinyl chloride or acetate to "anchor" the cellulose fibres, or the application of oiling during laundering. The cotton industry needs to face this challenge to discover processes which will cut down or eliminate the loss of dust and fibre from all the cotton garments, bedlinen and furnishings in the hospital wards. The effect of cellular weave of the cotton blanket may also provide a useful lead in this respect.

SUMMARY.

Air-borne cellulose fibres circulating in a hospital ward and sampled at bed height contain large numbers of viable organisms, including coagulase-positive antibiotic-resistant *Staph. aureus*. These bacteria-carrying fibres constitute an important potential source of wound contamination and of other hospital-acquired staphylococcal infections.

The treatment of cotton goods to ensure that contaminated dust and fibre are retained on them and not shed to the air demands urgent solution.

ACKNOWLEDGEMENTS.

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ORAL PREMEDICATION OF CHILDREN WITH TRIMEPRAZINE TARTRATE.

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MANY children object to injections, but unfortunately oral premedication is not completely satisfactory. The disadvantages of oral premedication are uncertainty of absorption and possibility of vomiting, less certain drying of secretions by atropine or hyoscine given by mouth, and occasional refusal by the child to swallow the medicament.

However, successful oral premedication results in a less suspicious and more cooperative child, and one who will more readily accept a thiopentone induction of anæsthesia. The advantages of oral premedication are especially evident if a group of children is to be premedicated. If one child cries or struggles and needs forceful restraint for an injection, the apprehension of the remaining children is increased.

We were, therefore, interested to gain experience with a new drug for oral premedication, trimeprazine tartrate. Trimeprazine tartrate ("Vallergan") is a phenothiazine derivative described as having pharmacological actions intermediate between those of promethazine ("Phenergan") and chlorpromazine ("Largactil"). It has marked sedative, anti-emetic and antihistaminic actions, and potentiates general anæsthetic agents. There is little sympatholytic, ganglion-blocking or hypotensive activity. When it is taken orally, there is an appreciable local anæsthetic action on the mucous membrane of the mouth.

Method of Trial.

At first the makers' recommendations of dose and time of administration were followed, and these factors were subsequently altered to suit our specific requirements. Atropine was given parenterally until we had sufficient experience with the trimeprazine, when a mixture of hyoscine hydrobromide and trimeprazine was tested. Dosage was by body weight, without regard to age, obesity or psychological make-up. Sedation was judged as "good" if the patient needed no, or minimal, reassurance, "fair" is reassurance was necessary and "poor" if the child could not be reassured.

The antisialogogue activity of the hyoscine given by mouth was classified as "adequate" or "inadequate". It was judged to be inadequate if secretions interfered with induction or maintenance of anæsthesia or necessitated frequent suction.

To simplify observations, the patients came from one ward and all operations were ear, nose and throat procedures. The method of induction of anæsthesia was inhalational or intravenous; it was maintained by nitrous oxide and oxygen, with or without ether given by a Boyle-Davis gag or an endotracheal tube.

Results.

Trimeprazine was administered on a total of 160 occasions. When the recommended dose of 1 mg. per pound

of body weight one hour before operation was used, it soon became apparent that unless the drug is given at least one and a half hours before operation there is inadequate absorption. This level of dosage was characterized by occasional pre-operative restlessness and, more commonly, restlessness of varying degree in the immediate post-operative period. In all cases, prolonged drowsiness, pallor and tachycardia were present.

The dose was then reduced to 0·8 mg. per pound of body weight. The same complications were seen, but to somewhat less degree. Post-operative tachycardia, although slightly less, was still troublesome, making difficult the recognition of post-operative hæmorrhage and pulmonary collapse. More satisfactory results were obtained with a dose of 0·5 mg. per pound of body weight, which is the dose one might expect of a drug so closely resembling chlorpromazine and promethazine. Pre-operative sedation was satisfactory, without any instances of pre-operative restlessness. Tachycardia and post-operative restlessness were less troublesome. On return to the ward from the recovery room, the patients were quiet and drowsy, but easily roused. We judged that, for our purpose, 0·5 mg. of trimeprazine per pound of body weight, given at least one and a half hours before operation, gave adequate sedation. This dose is conveniently prescribed as 1 dr. of the commercial syrup per stone of body weight, each drachm of "Vallergan" syrup containing 7·5 mg. of trimeprazine.

Hyoscine hydrobromide was then added to the syrup, at first in a ratio of one four hundred and fiftieth of a grain (0·14 mg.) per drachm. This dose produced adequate drying in only 85% of cases. Increasing the amount of hyoscine hydrobromide to one three hundred and sixtieth of a grain (0·18 mg.) per drachm produced adequate drying in 95% of cases. This is a dose ratio of 0·012 mg. of hyoscine hydrobromide per pound of body weight. No untoward reactions have been seen with this dosage. Post-operative tachycardia was further reduced and pre-operative sedation slightly improved. Post-operative restlessness was not more marked, nor was post-operative drowsiness increased. The dose of trimeprazine syrup with hyoscine was limited to a maximum of 6 dr.

The assessment of the pre-operative sedation by trimeprazine is summarized in Table I.

TABLE I.
Pre-Operative Assessment of Sedation by Trimeprazine Tartrate.

Dose per Pound of Body Weight.	Number of Cases.	Pre-Operative Sedation.		
		Good.	Fair.	Poor.
1·0 mg.	20	14	2	4
0·5 mg.	50	25	17	8
0·5 mg plus hyoscine hydrogen bromide, 0·012 mg.	40	24	12	4

In the total series there has not been a case of persistent post-operative vomiting, nor has any child refused the drug or vomited it before operation. There were two cases of post-operative pneumonia, both when the higher dosage of trimeprazine was used. No other complications were seen, either in the immediate post-operative period or later.

Early in the investigation it was obvious that the drug was most useful for children aged over three and a half years, and we soon abandoned its use for those below this age.

To assess the value of this new drug more accurately we are now carrying out a double-blind test to compare the effects of trimeprazine plus hyoscine, another sedative plus hyoscine, and hyoscine alone.

Summary.

Trimeprazine tartarate, 0·5 mg. per pound of body weight, plus hyoscine hydrobromide, 0·012 mg. per pound of body weight, given orally one and a half to three hours before operation, achieves good or fair sedation in 90% and adequate drying in 95% of children over the age of three and a half years. Children readily accept such a mixture, and post-operative vomiting is minimal.

Acknowledgements.

It is a pleasure to acknowledge the assistance and advice of Mr. S. A. Downie and Mr. G. Lee, of the Pharmacy Department, Adelaide Children's Hospital. We are grateful to May and Baker Ltd. for supplies of "Vallergan".

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Reports of Cases.

APLASTIC ANÆMIA AND AGRANULOCYTOSIS FOLLOWING CHLORPROMAZINE THERAPY.

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SINCE its introduction in 1952, chlorpromazine has been extensively used in both psychiatry and general medicine. Agranulocytosis following the use of this drug was first reported by Lomas in 1954. Further reports have appeared since, including one from Australia (Johnson, 1957), though in his case "Pacatal" had also been used. In spite of this growing literature, there still appears to be little awareness of this rare, but dangerous, complication. It is for this reason that this case is reported, and also because it shows the occurrence of depression of erythroid as well as myeloid elements of the bone marrow.

Clinical Record.

On June 17, 1958, a female patient, aged 35 years, was admitted to the Mercy Hospital, East Melbourne, for Cæsarean section. She was a dwarf with kyphoscoliosis, resulting from spinal tuberculosis, which had appeared at the age of four years. She spent much of the next ten years in hospital, but at the age of fourteen the lesion was regarded as healed. In spite of her small abdominal cavity, the growing fœtus had been remarkably well accommodated, particularly after it was turned to the transverse position at the twenty-fourth week. The pregnancy had been uneventful apart from mild hypertension, which was controlled by 0·75 mg. of reserpine given daily from the fourteenth to the thirty-sixth week. At that stage the patient's blood pressure rose suddenly to 190/120 mm. of mercury. Except that she refused to believe that she would ever be delivered of a live baby, the patient appeared to be mentally stable.

The Cæsarean section operation was uncomplicated, and the immediate post-operative period uneventful. However, it was difficult to convince the patient that her baby was alive. This position was aggravated by the fact that the baby was in an "Isolette" and could not be shown to her. Seven days after the operation the patient's behaviour became obviously abnormal. She became violent and abusive, talked incessantly, and insisted that the baby was dead. The intravenous injection of 0·1 gramme of azacyclonol hydrochloride ("Frenquel") having proved ineffective, she was treated with chlorpromazine ("Largactil"), 0·1 gramme being given intravenously at intervals of between eight and twelve hours. There was

marked improvement, and two days later oral chlorpromazine therapy at the same dosage was commenced. Later that day therapy was discontinued because of tachycardia (pulse rate 160 per minute) and a respiratory rate of 32 per minute. There was no fever or other evidence of infection. On the following day the pulse and respiratory rates had subsided, but the patient's mental condition was deteriorating. No improvement was produced by perphenazine ("Trilafon"), or further chlorpromazine, so electro-convulsive therapy with "Pentothal" and "Brevidil" anæsthesia was commenced. By July 6, after seven treatments, the patient's behaviour and mental state appeared normal. Two days later she was discharged home, and chlorpromazine was prescribed in doses of 0·1 gramme twice a day. This treatment was continued until July 28, 1958, when it was stopped after the development of fever and sore throat. The total dosage of drugs taken by the patient over this period of 32 days was as follows: 8·65 grammes of chlorpromazine, of which 0·65 gramme was given intravenously; 0·24 gramme of perphenazine; 0·1 gramme of azacyclonol; and 0·72 gramme of progesterone. Small amounts of stilbœstrol and chloral hydrate had also been given.

On July 29 the patient was admitted to the Box Hill and District Hospital. There was a vague history of a "chill" some two weeks previously, and of fever, sore throat, and pain in the right hand and axilla for three days. On examination of the patient, her temperature was found to be 104·6° F. and her pulse rate 180 per minute. There was marked pallor. On the right index finger there was an incised paronychia, with lymphangitis and large tender glands in the axilla. The pharynx was red and œdematous and covered with white patches resembling thrush. Examination of the blood on the following day showed 8·6 grammes of hæmoglobin per 100 ml. and 100 leucocytes per c.mm. Examination of blood film showed that the red cells were slightly hypochromic and microcytic. Of the leucocytes, 98% were lymphocytes and 2% monocytes. No cells of the granular series were seen. Platelets appeared to be in excess. Bone-marrow biopsy on the same day showed a gross depression of myeloid and erythroid cells.

Blood transfusion, and large doses of crystalline penicillin (12,000,000 units per day) and streptomycin (two grammes per day) appeared to produce some temporary clinical improvement; however, apart from the rise in hæmoglobin level due to transfusion, the blood picture was unchanged. Death occurred suddenly on August 3.

At autopsy the major branches of both pulmonary arteries were filled with recent ante-mortem thrombus. There was some congestion and ulceration of the trachea, bronchi, œsophagus and stomach. Bone marrow from the vertebral bodies and from the right iliac crest was pale and watery. On microscopic examination the spleen and the bone marrow showed atypical reticulum-cell hyperplasia. An accurate cell count on the marrow was difficult because of autolysis. However, it appeared that the proportion of cell types was approximately the same as in the biopsy specimen.

Conclusion.

It seems reasonable to assume that chlorpromazine was responsible for the bone-marrow aplasia in this case. Most reported cases of agranulocytosis associated with chlorpromazine therapy have been in the mentally ill, and it is probable that this is due to the large dosage used for such patients. Even then, however, it is a rare complication. Melrose (1958) estimates its occurrence at less than one in 50,000 patients taking the drug. The Mental Hygiene Department of Victoria has used chlorpromazine in large doses in many thousands of cases, and has not encountered agranulocytosis directly attributable to the drug (Stoller, 1959). A somewhat different experience is reported by Pisciotta *et alii* (1958), who found agranulocytosis in 16 patients out of a total of more than 3000 to whom this drug had been administered. This series was also of interest because in half their cases the condition appears to have been diagnosed by routine leucocyte counts prior to the onset of symptoms. Many authorities believe that regular leucocyte counts have little value in detecting the onset of agranulocytosis (Sturgis, 1955; Melrose, 1958). However, De Gruchy (1958) advises regular blood examination when "high risk" drugs are being used. Figures other than those of Pisciotta's group suggest that chlorpromazine is a "low risk" drug.

Awareness by the physician of the early symptoms, and instructions to the patients and their families of the steps to be taken should such symptoms develop, appear to be the most practical safeguard against marrow damage from this drug.

Acknowledgements.

We wish to thank Dr. Neil Carson, of Blackburn, Victoria, for clinical data concerning this patient, and Dr. M. K. Shoebridge for the pathological examinations.

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CORNEAL ŒDEMA DUE TO "CHLOROQUINE".

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"CHLOROQUINE" is a drug which, at first used as a suppressive in malaria, is now widely used in rheumatoid arthritis, amœbiasis, lupus erythematosus and actinic dermatitis. Since it has been used more extensively, reports are coming to hand of its toxic effects as they affect the eyes. Its most important ocular manifestation is œdema of the cornea with symptoms suggestive of glaucoma.

In 1946 Reese reported the cases of three patients who had been taking "Atebrin" in doses of 0·2 gramme daily and developed corneal œdema. Suspension of the drug led to clearing of the cornea, and relapse occurred on readministration. There was no inflammatory reaction or corneal staining with fluorescein, but the cornea showed an œdema of unusually fine texture and uniform appearance.

Chamberlain and Boles (1946) described similar findings in four patients who had been taking 0·1 gramme of "Atebrin" daily. Mann (1947) described six cases in which ocular symptoms developed—namely, blue haloes around lights. These patients worked in the production of "Atebrin", in an atmosphere contaminated by "Atebrin" dust. They had no corneal œdema, but in each case fine dust particles were present intracellularly in the corneal epithelium, and across the lower half of each cornea were wavy yellow lines. Removal from contact with "Atebrin" produced complete cure.

Calkins (1958) describes corneal œdema in seven patients who had been under treatment with "Chloroquine" for amœbiasis, rheumatoid arthritis, lymphocytoma cutis, lupus erythematosus and primary scleroderma. All complained of fuzziness of vision and haloes around lights. In the corneæ there were a diffuse, fine, droplet-like change and opacity limited to the epithelium and subepithelial layers, but not involving Bowman's membrane.

In cases of longer standing there was focal linear increase in the opacity, resembling the spokes of a wheel.

In a preliminary communication Hobbs and Calnan (1958) report the cases of 30 patients under treatment with "Chloroquine". Three of these patients only experienced spontaneous perception of haloes. Nineteen complained of visual upset, and of these 19 patients the corneæ were normal in four. In the nine patients who had no symptoms, all had changes in the corneal epithelium. The corneæ in these cases had a similar appearance to those described by Calkins (1958), but with some changes in the stroma beneath Bowman's membrane in a few cases. Hobbs and Calnan were able to conclude from their findings that a complaint of visual disturbance is an unreliable guide to the presence of corneal changes. The ocular symptoms were uniform in all the cases reported—blurred or misty vision, difficulty in driving a motor-car. In practically all cases visual acuity indoors as tested with Snellen type was normal, but vision was particularly affected at night, and those who drove a car had great difficulty.

Clinical Record.

A female patient, 61 years of age, was examined on April 28, 1959, with a complaint of foggy vision for distance present for about two and a half months. Indoors she was comfortable, but outdoors her vision was blurred and she was unable to drive her car. For a similar period she had noticed haloes around lights. The corrected visual acuity was 6/6 in each eye; the eyes were white, but both corneæ showed a gross diffuse corneal œdema reminescent of glaucoma. There was no staining with fluorescein. The ocular tension was 18 mm. of mercury (Schiötz) in each eye. Slit-lamp examination showed a fine, diffuse and uniform corneal œdema, and in the deeper epithelial layers below the pupil were linear striations almost star-like in pattern. A provisional diagnosis of corneal dystrophy was made, but as the appearance was not typical, contact was made with the patient's physician, who had been treating her for rheumatoid arthritis. The patient had commenced a course of "Chloroquine" on November 25, 1958, in a dosage of 100 mg. twice a day for two weeks, and since that time had been taking 200 mg. of "Chloroquine" twice daily. On April 30, 1959, the dose of "Chloroquine" was reduced to 100 mg. twice daily, and on May 5, 1959, "Chloroquine" therapy was suspended. On June 2, 1959, the patient was of the opinion that haloes were less troublesome, and her vision at night had improved. Examination showed that there was definite clearing of the cornea, but that the linear streaks were unchanged. On June 30, 1959, there was no evidence of corneal œdema in either eye, and the linear opacities were breaking up and disappearing. At a last examination on July 28, 1959, she was completely asymptomatic, and slit-lamp examination failed to reveal any abnormality in either eye.

Discussion.

With widespread use of "Chloroquine", it is probable that a certain number of patients will complain of visual upset.

Robinson (1959), in a personal communication, informed me that he had seen at least one other rheumatoid arthritis patient under treatment with "Chloroquine" with visual upset. In view of the findings of Hobbs and Calnan, it may be wise to have all patients on prolonged "Chloroquine" therapy examined at regular intervals with a corneal microscope.

The means by which "Chloroquine" produces corneal œdema is unknown; but it is not impossible that a very close liaison between physician and ophthalmologist may produce an answer.

Because of the widespread clinical value of "Chloroquine" in a variety of disease entities, there is no desire to cause undue alarm in its use in accepted dosage. Rather it is intended by the publication of these findings, which have been carefully checked, to encourage a specific search for corneal change in patients receiving the drug, and the identification of the substance in the cornea, in order that methods of controlling the corneal change may be developed while the patient continues the use of this important therapeutic agent (Calkins, 1958).

Acknowledgement.

I should like to thank Dr. R. G. Robinson for his notes on this patient and for his comments on the use of "Chloroquine" in rheumatoid arthritis.

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Reviews.

The Differential Diagnosis of Abdominal Pain. Edited by *Sherman M. Mellinkoff, M.D.; 1959. New York, Toronto* and London: McGraw-Hill Book Company, Limited. 8" × 5", pp. 458, with 28 illustrations. Price: $9.00.

THIS is the first of a number of projected volumes based on symposia held at the Medical Centre of the University of California (Los Angeles). Though it has a single editor its authorship is multiple, and this has resulted in some repetition and overlapping in the different sections. In general, however, it is a very good book, and serves to remind us that the radiologist and the biochemist now play parts in this field that were undreamt of in the years when most of the standard books on this subject were written.

The book lacks nothing in completeness, and every physical lesion that has ever been credited with producing abdominal pain receives some mention. However, the subject of emotionally induced pain receives much less generous measure. The subject is methodically developed, an initial chapter on basic considerations in the study of abdominal pain being followed by chapters on medical diseases, surgical diseases and retroperitoneal causes of abdominal pain, and a consideration of chronic or recurrent pain.

If the book has a weakness, it stems from the basic considerations. While the various tissues from which pain may be derived are carefully enumerated, and proper emphasis is given to the importance of the summation of subliminal stimuli, it is not always made clear whether the resultant pain is experienced in the affected organ, or is due to reference to more superficial structures, by a process of cerebral misinterpretation.

Insufficient importance is attributed to the depth of the stimulus from the body surface. From the clinical viewpoint, the mechanism of tenderness is at least as important as that of pain, and the failure to recognize its association with emotionally induced neuralgias of the abdominal wall must make for frequent diagnostic error. This is the more surprising, since the statement is made in italics that the assumption that, because pain is severe, it must be of organic rather than of functional origin, is dangerously misleading and has been the basis of many unfortunate instances of multiple laparotomy.

In the section on the pain of peptic ulcer, though it is recognized that the mechanism requires the contact of acid gastric juices with raw surfaces of live tissues in the ulcer base, and though the importance of associated inflammation is stressed, the author does not seem to recognize the extent to which periduodenal or perigastric spread of inflammation accounts for the symptoms of the ulcer attack.

The chapter on retroperitoneal causes of abdominal pain is excellent.

A short section on psychiatric disorders as causes of abdominal pain is weakened by the assumption that

abdominal pain of emotional origin can usually be recognized as the symptom of dysfunction in the intestinal tract. Surely only a small proportion of nervous abdominal disorders fall into this category.

Apart from the points mentioned, this book exhibits much wisdom, and we can only applaud such statements as the following:

> Long puzzling problems in abdominal pain are more often solved by taking another history than by any other technique.
>
> The radiologist may tell the patient's physician that a duodenal ulcer is present, but the physician must decide for himself what is causing the patient's abdominal pain.

No practising doctor could fail to gain from reading this book, and in many respects it is the best exposition of the subject known to us.

Dangerous Marine Animals. By Bruce W. Halstead, M.D.; 1959. Cambridge and Maryland: Cornell Maritime Press. 9" x 5¾", pp. 160, with 86 illustrations. Price: 30s. (English).

DR. BRUCE HALSTEAD, formerly Instructor in Tropical Medicine in the United States Naval Medical School, after fourteen years of study and research on dangerous marine animals has gathered together sufficient material for this admirable book, aimed to enlighten and warn the intelligent lay reader. All the marine animals which can bite or sting or be poisonous as food are here described, with an easily understood account of the signs and symptoms they produce in the human victim and with guides for first-aid treatment. Some Australians may resent omission of their country from the lists of sites where such dangerous creatures are to be found; but though the book has been written by an American author for American readers, actually the information given is fully applicable to Australia. Warm tributes are paid to Dr. Gilbert Whitley and Dr. V. M. Coppleson, and two of the excellent illustrations have been supplied by the Townsville ichthyologist, Mr. George Coates.

The illustrations are throughout of high standard, and the letterpress is commendably clear and accurate. Needless to say, the presentation of medical matter is reliable. A comprehensive and well-documented treatise on toxic marine organisms of scientific standard is promised, and if this preliminary volume can be taken as an indication of the author's capacity, such a work will indeed be welcome.

Clinical Obstetrics and Gynecology. Volume 2, Number 1: March; 1959. "Spontaneous Abortion", edited by David N. Danforth, M.D.; "Menstrual Disorders", edited by C. Frederic Fluhmann, M.D. New York: Paul B. Hoeber, Inc. 9¼" x 5¾", pp. 252, with illustrations. Price: $18.00 per year.

IN opening the symposium on spontaneous abortion, Wilson and Smith of the Mayo Clinic show the fallacy in the belief that trauma, travel, operations or emotional upsets play any significant part in the causation of abortion.

Warburton and Fraser write from McGill University on genetic changes which can theoretically cause abortion. They append a long list of references to world literature. Corner of John Hopkins University advocates the use of progesterone in adequate amounts for threatened abortion, and gives a warning against the androgenic effect of some recent synthetic progestins. Danforth writes a lucid account of the anatomy, diagnosis and treatment by suture of the incompetent cervix responsible for mid-trimester miscarriage. (Barter discussed the technique of this procedure in the previous number of this series.)

MacLeod (Cornell) sums up as "not proven" the implication of spermatozoa in the ætiology of abortion.

The diagnosis and treatment of threatened and of habitual abortion are discussed by Haskell (University of Utah). He points out the illogicality of prolonged bed rest for patients whose bleeding does not cease in 24 hours, and gives an appraisal of oral progestin administration.

D'Esopo describes the routine management of abortion at the Sloane Hospital, New York. Chloramphenicol is given by intravenous infusion before curettage in cases of infection. The fibrinogen levels are closely watched in cases of long-standing dead ovum. It is interesting to note that of 150 cases of missed abortion quoted by Cosgrove of the Margaret Hague Hospital, in only one was there an abnormal clotting mechanism, and this occurred after a massive hæmorrhage following untimely dilatation and curettage.

Mulé and McCall, with a wide experience in New Orleans, where in 20% of abortion cases sepsis is present, belong to the conservative school, and except in hæmorrhage withhold curettage until several days after the temperature has returned to normal. They stress the significance of infection and the high mortality rate (2·4%).

McElin describes the therapeutic abortion committee as set up in the Evanston Hospital. There is undoubted value in such a system.

In the symposium on menstrual disorders an excellent paper on premenstrual tension is given by Joseph H. Morton. The logical approach to therapy is discussed in detail, and an extensive bibliography is appended. The longest chapter in this number, and one of the most interesting, is by Allan Palmer, on the use of the basal body temperature chart in the practical management of menstrual disorders. He demonstrates how pregnancy can be diagnosed earlier by this than by any other means, and how chorionic gonadotropin injections can lengthen the short post-ovulatory phase ("hiphase") associated with sterile cycles. Henriksen writes on the widespread incidence of *mittelschmerz*. The ætiology remains obscure. Irving Stein, Senior, of Chicago, gives a full account of the Stein-Leventhal syndrome. McLennan of Stanford University discusses dysfunctional uterine bleeding, especially endometrial hyperplasia and irregular shedding; while the hormonal control of these conditions is dealt with by Greenblatt (Augusta). An excellent account is given, with illustrations, of the proper use of progesterone and the newer progestational agents. These recent drugs are also surveyed by Holmstrom, who wrote the chapter on dysfunctional bleeding in Volume I, Number I.

Again we have no hesitation in recommending this series to all obstetricians and gynæcologists.

The Innervation of Muscle: A Biopsy Study. By C. Coërs and A. L. Woolf, M.D.; 1959. Oxford: Blackwell Scientific Publications. 9¾" x 7¼", pp. 168, with 280 illustrations. Price: 42s. (Abroad).

THE pathologist often feels that he should get more information from muscle biopsies than he actually does. The new techniques described in this book will go a long way to relieve the situation, and we must no longer regard intramuscular nerves and their endings as invisible agents. Although we cannot make a diagnosis by examining the nerve endings, the authors of this monograph show us how to recognize patterns in motor innervation corresponding to a considerable degree with particular diseases of the lower motor neuron or of the non-neural components of muscle. The state of the terminal portion of the axon and its endings can also give valuable prognostic information.

It is axiomatic that today's research method is tomorrow's routine. Therefore, it can be safely predicted that before long the vital staining of muscle by methylene blue and the demonstration of cholinesterase in the subneural apparatuses will be routine procedures in the investigations of neuro-myopathies and neuritides.

The illustrations of terminal axons and nerve endings are excellent, and many of the photomicrographs surpass even the idealized drawings of an older generation which relied chiefly upon Bielchovsky.

This book is for all who are interested in the innervation of muscle. The anatomist and physiologist as well as the pathologist and clinician will find it stimulating and useful.

Heredity of the Blood Groups. By Alexander S. Wiener, A.B., M.D., F.R.C.P., F.A.C.P., and Irving B. Wexler, A.B., M.D., F.A.C.P.; 1958. New York and London: Grune and Stratton, Incorporated. 8½" x 5½", pp. 160, with 4 figures and 51 tables. Price: $6.00.

MANY books have recently been published on the human blood groups, but this is the first which has been devoted solely to the mechanisms of their inheritance. Serological studies of families have contributed much to our knowledge of the blood groups, and it is logical that the authors should base their discussions largely on these studies. The introduction contains a long and valuable table of the history of discovery and of the genetics of the various groups. Succeeding chapters deal separately with each of the blood-group systems and the book concludes with some notes on the medico-legal and anthropological applications. The authors, especially the senior, have

published numerous papers on the inheritance of blood groups, and the book is really a summary of these papers.

The accuracy of the historical and serological facts as presented cannot be questioned, but many workers will find some of the authors' interpretations difficult to accept. Their theories of the nature of the agglutinogens and their genetic interpretations of the phenotypes are often ingenious and original, and should at least be considered by all workers. Unfortunately the authors sometimes tend to confuse fact with theory, and as a result many of their claims of proof are unconvincing. The views of the British workers have not been presented with complete impartiality, and many will regret that the controversy over Rh nomenclature is continued with unabated ferocity. Nevertheless, the book is lucidly written and should be read by all interested in human genetics.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"Psychiatry in the Medical Specialties", by Flanders Dunbar, M.D., Med.Sc.D., Ph.D.; 1959. New York, Toronto and London: McGraw-Hill Book Company, Incorporated. Published in cooperation with Landsberger Medical Books, Incorporated. 9" x 5¼", pp. 543. Price: $12.00.

"A Guide to the Identification of the Genera of Bacteria: With Methods and Digests of Generic Characteristics", by V. B. D. Skerman; 1959. Baltimore: The Williams & Wilkins Company. Sydney: Angus & Robertson, Limited. 9" x 5¾", pp 230, with illustrations. Price: £3 0s. 6d.

"Kurzer Atlas für das klinische Laboratorium", von Dr. med. Eberhard Goetze; 1959. Jena: Veb Gustav Fischer Verlag. Sydney: Angus & Robertson, Limited. 9¼" x 6¼", pp. 40, with many illustrations. Price: DM 12.75.

"Psychoanalysis of Today", by S. Nacht, American adaptation prepared by Ruth Emma Roman; 1959. London and New York: Grune & Stratton, Incorporated. 8¼" x 5", pp. 240. Price: $5.75.

"Rheumatic Fever, Epidemiology and Prevention: The Proceedings of a Seminar Held at the International Children's Centre, Paris, 25-27 September, 1956", edited by R. Cruickshank, M.D., Ch.B., F.R.C.P., D.P.H., and A. A. Glynn, M.B., B.S., M.R.C.P.; 1959. Oxford: Blackwell Scientific Publications. 8¼" x 5¼", pp. 194, with many tables. Price: £1 5s. (English).

"History of the American Dietetic Association, 1917-1959", edited by Mary I. Barber; 1959. Philadelphia and Montreal: J. B. Lippincott Company. Sydney: Angus & Robertson, Limited. 8" x 5", pp. 336, with 46 illustrations. Price: 66s.

"The Clinical Syndrome of Diabetes Mellitus", by John Lister, M.A., M.D., M.R.C.P. (Lond.); 1959. London: H. K. Lewis & Company Limited. 8¼" x 5¼", pp 244, with 34 illustrations. Price: £1 15s. (English).

"Smoking, the Cancer Controversy: Some Attempts to Assess the Evidence", by Sir Ronald A. Fisher, Sc.D., F.R.S.; 1959. Edinburgh and London: Oliver and Boyd. Adelaide: Rigby Limited. 8¼" x 5¼", pp. 48. Price: 2s. 6d.

"Cancer Current Literature Index", under the general editorship of W. van Westering; 1959. Amsterdam, New York: Excerpta Medica Foundation. 9¼" x 6¼", pp. 38. Price: $7.50 per year (subscription).

"Heroic Sanctity and Insanity: An Introduction to the Spiritual Life and Mental Hygiene", by Thomas Verner Moore; 1959. New York and London: Grune and Stratton, Incorporated. 8¼" x 5¼", pp. 256. Price: $5.00.

"Medicine Man", by Frank McCann; Illustrations by Phil Taylor; 1959. Sydney: Angus & Robertson, Limited. 7¾" x 5", pp. 216, with many illustrations. Price: 17s. 6d.

"Work and the Heart: Transactions of the First Wisconsin Conference on Work and the Heart", edited by Francis F. Rosenbaum, M.D., and Elston L. Belknap, M.D.; 1959. New York: Paul B. Hoeber, Inc. 10¼" x 7", pp. 560, with many illustrations and tables. Price: $12.00.

"Gynecologic Endocrinology", by Gardner M. Riley, Ph.D., with a foreword by Norman F. Miller, M.D.; 1959. New York: A. Hoeber-Harper Book. 9¼" x 6", pp. 350, with 67 illustrations and 16 tables. Price: $6.50.

"A Review of the Literature on the Distribution and Epidemiology of Filariasis in the South Pacific Region", by M. O. T. Iyengar; 1959. South Pacific Commission Technical Paper, No. 126. Noumea: South Pacific Commission. 10" x 8", pp. 172. Price: 6s. (sterling).

"Atlas of Tumor Pathology. Section VII: Fascicles 27 and 28: Tumors of the Pancreas", by Virginia Kneeland Frantz, M.D.; 1959. Published by the Armed Forces Institute of Pathology. 10¼" x 7½", pp. 152, with 94 illustrations. Price: $1.50.

"Pediatric Pathology", by Daniel Stowens, M.D.; 1959. Baltimore: The Williams & Wilkins Company. Sydney: Angus & Robertson, Limited. 10¼" x 7½", pp. 684, with 374 illustrations. Price: £11.

"Psychopathy: A Comparative Analysis of Clinical Pictures", by Carl Frankenstein, Ph.D.; 1959. New York and London: Grune & Stratton, Inc. 9" x 5¾", pp. 208. Price: $6.75.

"Midwifery (A Textbook for Pupil Midwives)", by Gordon W. Garland, M.D. (London), F.R.C.O.G., and Rosemary C. Perkes, S.R.N., S.C.M., M.T.D.; 1959. London: The English Universities Press, Limited. Melbourne: G. Malcolm Titt. 8¼" x 5", pp. 330. Price: 39s. 6d.

"Radiation Biology of Vicia Faba in Relation to the General Problem", by John Read, B.Sc., Ph.D.; 1959. Oxford: Blackwell Scientific Publications. 8¾" x 5½", pp. 292, with 74 illustrations and 41 tables. Price: 45s. (English).

"Medical Radiographic Technic", prepared by Technical Service X-Ray Department General Electric Company under the original editorial supervision of the late Glenn W. Files; revision by William L. Bloom, Jr., John L. Hollenbach, R.T., James A. Morgan, R.T., and John B. Thomas, R.T.; second edition; 1959. Oxford: Blackwell Scientific Publications. 10" x 7", pp. 400, with many illustrations. Price: 82s. 6d. (English).

"Textbook of British Surgery", edited by Sir Henry Souttar, C.B.E., D.M. (Oxon.), F.R.C.S., and J. C. Goligher, Ch.M. (Edin.), F.R.C.S. (Edin. and Eng.); Volume 4; 1959. London: William Heinemann, Medical Books, Limited. 9¾" x 7", pp. 708, with 405 illustrations. Price: £5 5s. (English).

"The Child with Abdominal Pains", by John Apley, M.D., F.R.C.P.; 1959. Oxford: Blackwell Scientific Publications. 8¾" x 5½", pp. 96. Price: 12s. 6d. (English).

"Treatment of Cancer in Clinical Practice", edited by Peter B. Kunkler, M.A., M.D. (Cantab.), M.R.C.P., F.F.R., and Anthony J. H. Rains, M.S. (Lond.), F.R.C.S.; 1959. Edinburgh and London: E. & S. Livingstone Limited. 9¾" x 6½", pp. 840, with many illustrations. Price: £5. (English).

"Growth Diagnosis: Selected Methods for Interpreting and Predicting Physical Development from One Year to Maturity", by Liona M. Bayer and Nancy Bayley; 1959. Illinois: The University of Chicago Press. 11" x 8½", pp. 256, with 171 illustrations and many tables. Price: $10.00.

"Biophysical Science—A Study Program", planned and edited by J. L. Oncley, editor-in-chief; F. O. Schmitt, R. C. Williams, M. D. Rosenberg and R. H. Bolt; 1959. New York: John Wiley & Sons, Inc.; 10¼" x 7½", pp. 138, with many illustrations and tables. Price: $6.50.

"Mental Symptoms in Temporal Lobe Epilepsy and Temporal Lobe Gliomas: With Special Reference to Laterality of Lesion and the Relationship between Handedness and Brainedness", by Torsten Bingley; 1958. Acta Psychiatrica et Neurologica, Supplement 120, Vol. 33. 9½" x 6¼", pp. 164, with 15 tables. Price: not stated.

"Miscellaneous Notes (Fourth Series)", by F. Parkes Weber, M.D., F.R.C.P., F.S.A.; 1959. London: H. K. Lewis & Company, Limited. 7½" x 4¾", pp. 32. Price: 6s. (English).

"Epidemic Diseases", by A. H. Gale; 1959. Mitcham, Victoria: Penguin Books Pty. Ltd. 7¼" x 4¼", pp. 160, with 21 illustrations. Price: 5s. 6d.

"Science News", Number 53, edited by Archie and Nan Clow; 1959. Mitcham, Victoria: Penguin Books, Limited. 7¼" x 4¼", pp. 128, with many illustrations. Price: 4s.

"Pharmacopœia Internationalis", First Edition, Supplement; 1959. Geneva: World Health Organization. 9½" x 6¼", pp. 244, with many tables. Price: £1 5s. (sterling).

"The Complete Cookery Book for Diabetics", by Iris Holland Rogers; second edition; 1959. London: H. K. Lewis & Company, Limited. 8¼" x 5¼", pp. 140, with 23 illustrations. Price: 6s. (English).

The Medical Journal of Australia

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THE WORLD'S HEALTH.

WITH the passing of each year more and more remarkable things are being done for the first time. The most spectacular and the most widely acclaimed have been achievements of the physical sciences, the release and development of atomic power, the launching of satellites, the sending of a rocket to the moon, greatly increased speed of practical air travel and so on. In the medical sciences remarkable things have also been done, including achievements in surgery of the cardio-vascular and respiratory systems and neurosurgery, in the control of infectious diseases, in the understanding and management of endocrine and metabolic disorders. A catalogue of recent first achievements of major significance could go on for a long time. One such achievement, however, which saw the light of day in 1959 and is on a world scale, has not attracted the notice it deserves. This is the first comprehensive report ever prepared on the world health situation.[1] Though not exhaustive, it provides a valuable picture of various aspects of the health of 157 territories and, in synthesis, delineates the broader world picture.

Under article 61 of the Constitution of the World Health Organization, Member States accept an obligation to report on the progress made towards the achievement of health. This obligation has remained in abeyance for various reasons during the early years of WHO, but in 1958 the eleventh World Health Assembly affirmed the responsibility of WHO to review the health situation in the world and confirmed that this was another essential function of the organization. The result is the report under discussion.

The main part of the report is taken up by accounts of the health situation in their own countries prepared by the individual Member States. These include information on the numbers of births, on general mortality and on infant and maternal mortality, and on the medical and health personnel employed in national services, in hospital services and in private practice. Each country's account is preceded by a brief general summary of the national situation designed to set the stage for a review of the health situation. These individual accounts are of the utmost importance, as each country has its own circumstances and problems, its own responsibilities and, to some extent, its own answers. These it must present in its own way if the over-all picture is to be accurate and useful. The Director-General of WHO, Dr. M. G. Candau, remarks in the preface:

> For an international organization like WHO one of the methods of serving Member States is to present faithfully in clear, simple outline a picture of the state of health of each country and territory, based on the information at present available, and to focus the spotlight with the correct degree of intensity on new developments. By this means each country could keep the world informed of its proposals and undertakings, and so all the peoples would become more familiar with one another's problems in an intelligible relation to resources of personnel, money, and equipment. Furthermore, the world would discover what is being done in a great endeavour to improve health on a global scale.

For these reasons Dr. Candau asks the national health authorities, in reading the report, to direct their attention especially to the efforts which are being made, often against mighty odds, to promote the health and welfare of peoples who were previously little known to them—a request which needs to be heeded not just by administrative health officials but by all who have any real concern for the health of mankind. At the same time the story of one country's struggles and achievements and even of its failures is an important part of the common knowledge, and may well be, as Dr. Candau states, a source of great encouragement to other countries on the same path of progress.

Part I of this report is made up of a "general survey", which takes into consideration all cultural, social and economic aspects affecting the health of the population. The seven chapters of this survey are concerned respectively with influences affecting the health of the peoples, the state of the public health, administration of health services and activities, the distribution of functions in the health services, institutions and field establishments, education and training, and future developments. It is pointed out that in a general way, today's distribution of population over the earth's surface, favoured as it is in the temperate zones and somewhat hampered in the tropics, suggests a strong link between health and geographical factors. On the other hand, modern techniques are modifying these factors to an increasing extent. Thanks to the increase in scientific knowledge and its application, the chain of influence between climate and health can now be broken at almost any point. Modern transport makes it possible to feed men in barren areas and to furnish them with dwellings comfortable in either glacial or torrid zones. New methods of medical care are protecting human beings to an increasing degree against the various organisms to which they have been a natural prey. Moreover, the greatest hindrances to social progress—disease, ignorance and poverty—are being attacked by governments throughout the world with increasing vigour. The statement is made that when conditions are estimated by the usual indices it is apparent that many of the diseases that cause ill health, misery and economic loss are already coming under control.

One of the most encouraging features of the world health situation today, according to this report, is that the nations are becoming increasingly conscious of the importance of public health as a factor in the social and economic development of a country and of the need for further progress in this direction. Just as govern-

[1] "First Report on the World Health Situation, 1954-1956", Official Records of the World Health Organization, No. 94; 1959. Geneva: World Health Organization. 11" × 8½", pp. 398, with 8 illustrations. Price: 17s. 6d.

ments are recognizing their growing responsibility for providing health services, the report continues, so the people are becoming aware of the need for their own participation in the endeavour to build up the health of the nation. "The truth has been realized that health cannot be imposed: its promotion requires team-work within the community. Many of the more recent trends in public health administration stem from this concept, and future developments will be governed by it." Amongst the subjects of particular importance in the future are further reductions in infant mortality, prevention of chronic degenerative diseases, prevention of dental caries, development of rest and rehabilitation methods and facilities, greater stress on the value of medical statistics, study of the health aspects of radiation, provision of an adequate number of doctors and encouragement of international cooperation. In the still underdeveloped communities continued stress will need to be placed on full control of infectious diseases, the development of rural health services and the improvement of living and working environment down to the level of even elementary sanitation in some places.

The reduction of infant mortality remains an important goal everywhere. In many parts of the world it has been achieved in notable degree; but in some countries it has been found that, although infant lives were saved during the first twelve months, half the children died before reaching the age of five years. According to the report, the main cause was defective nutrition, particularly lack of protein, with infectious diseases and faulty environmental sanitation as contributing factors. These facts, the report points out, indicate the need to extend welfare services to children of pre-school and school age, and to lay special emphasis on nutrition, health education and the prevention of infection. "Sound teaching of this kind in the schools is carried home by the children and often has the effect of moving the parents to pay more attention to the living and working environment." Stressing the value of health statistics, the introduction to the report asserts that preventive medicine without records is like a ship without a compass. Statistics need to be constantly evaluated to find out whether they are in fact leading us forward or merely providing routine figures for general administrative purposes, but without them it is impossible to assess the extent or gravity of the problem of disease. Some less advanced countries can still not produce such data, and this leaves important questions unanswered. As the report puts it, sickness and disability may have a far more profound effect than is at present realized, and it may well be that through the lack of accurate information on morbidity an intolerable burden of suffering, economic loss and social misery is imposed on the community. All countries thus need to be encouraged to collect adequate health statistics and consider them in relation to the broader international picture. In this, as in so much else in the field of health, an international outlook is important. The obligation still remains with the more advanced and economically more secure countries, to help those less favoured, but this is not enough and certainly should not be considered by either party as a form of patronage. Preventive medicine, the report states, is entering a new era—the era of the universal participation of the people, and there is great scope for development. However, the essential condition for any advance in health work is a modest and cooperative approach, for every country has both something to learn and something to teach. "Indeed, this principle of give-and-take is the very keystone of future progress." It cannot be too strongly emphasized that this principle applies to every country, irrespective of its development, wealth or past record, if we are to enjoy the unquestionable benefits of an international approach to the world's health.

Current Comment.

THE EFFECT OF VACCINE ON CANCER PATIENTS.

THE idea of attempting to influence the course of cancer by the use of autogenous vaccines prepared from the patient's own tumour is not new. At the beginning of this century Jensen observed that a transplantable mouse tumour could be caused to regress and disappear if treated with immune rabbit serum. Since then, numerous workers have experimented with the idea; some tried it on human subjects and some encouraging results were obtained, but these were never conclusive enough to inspire a vigorous prosecution of the subject, and in the light of the concepts current at the time it seemed inherently improbable that an organism could be conditioned to destroy what was in fact a part of itself. The subject appeared to be of mainly theoretical interest. However, the past decade has seen a revolutionary development of our knowledge of the auto-immune processes, which are now coming to be accepted as playing an important role in a number of hitherto obscure diseases. It was natural that this, coupled with new refinements of technique, should lead to a re-appraisal of the possibilities of enlisting auto-immune mechanisms against disseminated cancer. J. B. and R. M. Graham of the Roswell Park Memorial Institute, Buffalo, New York, have done precisely this, and a recently published report[1] sets out their preliminary results, together with a lucid discussion of the problems involved. The essential problem is of course this: the patient is threatened by a cancer whose presence does not evoke any effective response on the part of the patient's defence mechanisms; is there any way in which these mechanisms can be stimulated into action? As the Grahams put it, "it is evident that one must offer either a more antigenic preparation of the tumour or change the patient so that he is more reactive". It is in this context that the researches in recent years of Witebsky and other workers into the experimental production of auto-immune reactions is particularly significant. It is well known to workers on immunology that the immune response of an organism to various substances can be greatly intensified by the simultaneous administration of various adjuvants. One of the best known of these, widely used in such investigations, is the Freund adjuvant, which consists of a light mineral oil, an emulsifying agent, and killed mycobacteria. The technique is well established of using such adjuvants to develop an immune response by the organism against its own tissues (e.g. nerve, testis or thyroid), when extracts of these are simultaneously injected. The Grahams used Freund adjuvant or one of its modifications in combination with extracts of the patient's own tumour in an attempt to elicit an immune response against the tumour tissue. One hundred and fourteen patients with advanced cancer, mostly patients with cancers of the uterus or ovaries, were treated by this method. Thirteen of these died within one month of starting treatment. The remainder had been treated with four different types of tumour extract, administered according to various schedules. Some had undergone prior operations; some received subsequent treatment in the form of palliative radio-

[1] *Surg. Gynec. Obstet.*, 1959, 109: 131 (August).

therapy, full radiotherapy, or chemotherapy. It is therefore difficult to assess the exact significance of the results obtained. The Grahams limit themselves to stating: "It is our impression that some of the patients have been benefited by the vaccine." Fourteen of the original 114 patients were alive and apparently free from disease at the time of writing of this report, seven to 30 months after receiving treatment. Some of these would have been expected to achieve this result in any case, in view of other treatment received, but in two of these patients complete excision of the original tumour had been impossible, and no other treatment had been received apart from four injections of adjuvant and tumour preparation.

However, certain valuable information was obtained. At the beginning of the study there was "grave concern" as to whether the Freund adjuvant could be given without serious risk to the patient of auto-immune reactions of undesirable types. In the event, no such untoward reactions were noted, though some patients have been under observation for nearly three years after their original treatment. Various technical difficulties had to be overcome. In the latter part of their series the Grahams made no attempt to render their tumour preparations bacteriologically sterile, though the source material was often grossly infected; yet no untoward reactions and no infections developed. It was found surprisingly difficult to obtain sufficient tumour material for preparation of the vaccine; in only a minority was the cancer found in large enough masses to provide five grammes of relatively pure tumour. This report throws new light on an intriguing and difficult problem, and clears the way for a more exact appraisal of the possible value of the techniques which have been elaborated. It appears to be a useful advance in one sector of a very large front.

BLOOD GROUPS AND DISEASE.

IN the last few years a considerable amount of work has been done on the relationship between blood groups and the incidence of certain diseases. The chief problem encountered in this type of investigation has been the difficulty of obtaining adequate samples of patients suffering from the disease under investigation and sufficient numbers of comparable controls. In spite of the inherent difficulties in such investigations, and in spite of criticisms that have been made of some of the statistical methods used, most workers believe that there is, in fact, a relationship between blood groups and the occurrence of certain diseases. The examples in which the evidence for such relationships is strongest are in increased incidence of Group O among patients with duodenal and gastric ulcer and an increased incidence of Group A among patients with carcinoma of the stomach and patients with carcinoma of the uterine cervix.

Now Hembold[1] has put forward a theory as to a possible mechanism by which blood groups can influence the development of disease. Considering carcinoma first, he suggests that when carcinoma cells develop in the body they may be sufficiently "foreign" to give rise to antibody production. However, if the patient belongs to Group A, the carcinoma cells, like the rest of the cells in the body, will contain part of the mosaic of the A antigen. Consequently, the antibody produced against the carcinoma cells will have a certain amount of specificity for the A antigen and will be taken up by that antigen in the normal body cells before it can destroy the carcinoma cells. On the other hand, if the patient belongs to Group O, when antibody is formed against the carcinoma cells, there will be nothing in its structure to react with normal body cells and it will remain free to combine with and destroy the carcinoma cells. Therefore carcinoma is less likely to develop in Group O individuals.

One of the difficulties in proving this theory is that the postulated mechanism, if it exists, acts at a time before a carcinoma has become established and made its presence known and therefore the hypothesis cannot be readily proved by experiment. Hembold suggests a number of lines along which research might be directed to elucidate the problem.

The mechanism suggested in peptic ulcer is slightly different. Here the antigen-antibody reaction is believed to take place locally at the ulcer site. In Group A people, after an initial injury to the mucosa, which might arise from any one of several causes, the damaged tissue is thought to give rise to an antibody with some specificity against the A antigen. This antibody, as in the suggested mechanism operating in carcinoma, will be absorbed by the A antigen on the body cells. In Group O people, on the other hand, the damaged tissues will produce an antibody with no such component to combine with the general body cells. Therefore, this antibody will be free to act on the damaged tissue at the site of the lesion. So when the gastric or duodenal mucosa is damaged from any cause, the situation will be that in an A person the mucosa will probably heal, whereas in an O person the damage will tend to be aggravated by a local antigen-antibody reaction and the ulceration will be likely to become chronic.

It is of interest that this theory is purely immunological, and Hembold does not invoke any genetic mechanism, such as genetic linkage, to explain these relationships. It provides a possible explanation of observations that have been confirmed by many groups of workers and are now widely accepted. Whether true or false it should provide a stimulus to further research along some interesting lines.

[1] *Blut*, 1959, 5: 7 (February).

DIABETIC NEUROPATHY.

WITH increasing life expectancy, diabetics are more liable to be afflicted with neuropathy, nephropathy, retinopathy, and peripheral vascular disease. In a recently published monograph Sven-Erik Fagerberg[1] has analysed the complications seen in 356 diabetic patients of whom all but 53 were examined by him personally. He found that neuropathy was, as a rule, absent only in those whose diabetic state had been of short duration. The incidence of retinopathy, nephropathy and peripheral vascular disease was higher in diabetics with neuropathy than in diabetics without neuropathy, and this higher incidence was statistically significant. Whereas peripheral vascular occlusive disease is four or five times more common in men than women amongst non-diabetics, the incidence is equal in diabetics. Diabetic retinopathy and nephropathy are based upon vascular changes. Therefore, considering these facts, viz. that these complications are dependent upon vascular changes and that they are more common in patients with neuropathy, it seems reasonable to suppose that the neuropathy also is based upon vascular alterations.

Accordingly Fagerberg has endeavoured to prove this hypothesis by correlating myelin sheath degeneration and vascular abnormalities in specimens of sural nerve obtained by biopsy or at autopsy. There was good agreement between myelin sheath degeneration and clinical signs of neuropathy. It was found that intraneural vascular lesions in the form of hyalinization, calibre reduction and wall thickening were more common in diabetics with neuropathy than in diabetics without neuropathy or in non-diabetic hypertensives. There was in addition, strong PAS-positive staining in severely affected vessels of diabetics but not in those of non-diabetics. While the myelin changes in the sural nerve in diabetic neuropathy cannot be definitely ascribed to vascular changes, there is a suggestion that there may be an alteration in the ground substance which is specific for diabetes.

[1] "Diabetic Neuropathy: A Clinical and Histological Study on the Significance of Vascular Affections", by Sven-Erik Fagerberg; 1959. Acta Medica Scandinavica, Supplement 345. Stockholm: Acta Medica Scandinavica. 9½" × 7", pp. 100, with 27 illustrations. Price not stated.

Abstracts from Medical Literature.

OPHTHALMOLOGY.

Unilateral Exophthalmos from Intracranial Lesions.

G. S. CAMPION (*Amer. J. Ophthal.*, June, 1959) states that exophthalmos may be the result of endocrine disease, orbital disease or intracranial disease. Of the intracranial causes of unilateral exophthalmos, meningiomas are the most important. Next most common are gliomas of the optic nerve or chiasma. Arterio-venous aneurysm between internal carotid artery and cavernous sinus is also a fairly common cause. The author discusses meningiomas as a cause of unilateral exophthalmos.

Ocular Manifestations of Occlusive Arterial Disease.

R. H. MINOR *et alii* (*A.M.A. Arch. Ophthal.*, July, 1959) discuss the ocular signs of occlusive disease of the vertebral-basilar arterial system, on the basis of 183 cases seen at the Mayo Clinic. Some ocular abnormality was present in 144 of the patients. The findings included homonymous hemianopia, transient blurring of vision, transient periods of total visual loss, and transient visual field defects. Other defects noted were diplopia, nystagmus, internuclear ophthalmoplegia, paresis of conjugate gaze, ptosis, distorted vision and conjugate deviation. The development of bilateral homonymous hemianopia and internuclear ophthalmoplegia appears to be diagnostic of basilar artery occlusion. Anticoagulant therapy was effective in checking further progression of the occlusive processes and in preventing further episodes of cerebrovascular insufficiency or thrombosis, but was not effective in restoring neurological function that already had been lost.

Combination Operation for Cataract with Glaucoma.

W. L. HUGHES (*Amer. J. Ophthal.*, July, 1959) describes an operation, comprising anterior sclerectomy, iris inclusion and lens extraction, for use in patients who have glaucoma and cataract. He has used the operation successfully in over 60 cases. Only 40 patients required miotics post-operatively to control tension. The author describes his technique in detail.

The Treatment of Iris Melanoma.

A. B. REESE AND G. W. CLEASBY (*Amer. J. Ophthal.*, May, 1959) review 157 cases of iris melanoma. The patients fall into three groups, those who had excisional iridectomy, those who had enucleation, and those who were observed. Indications for iridectomy are as follows: restriction of the tumour to the iris, the presence of established growths or signs of active change, the absence of tumour seedlings, the absence of glaucoma, and the presence of the suspected tumour near but not involving the ciliary angle; if the suspected tumour is situated towards the pupil margin, it may be observed, but if it is near the periphery of the iris, further growth would involve the angle and iridectomy is indicated. Excessive apprehension of the patient may be sufficient to warrant iridectomy. Enucleation is indicated when the tumour is not sufficiently localized for removal by iridectomy and when it is known that tumour tissue remains in the eye after iridectomy. In 140 cases in which follow up was possible there have been four deaths due to metastatic spread. The author outlines his technique for excisional iridectomy.

Tonography in Early Glaucoma.

W. ROBERTS (*Amer. J. Ophthal.*, July, 1959) conducted a survey to detect early glaucoma. Methods of examination included tonometry, visual field study, gonioscopy, provocative tests and tonography. The positive diagnosis of simple glaucoma in many cases was made on the basis of tonographic critera. From this study the author is of the opinion that, in the control of primary glaucoma, the aim should be to reestablish normal facility of outflow. He also considers that tonography is an essential examination in the diagnosis and treatment of glaucoma.

Applanation Tonometry in the Diagnosis and Treatment of Glaucoma.

B. BECKER AND A. GAY (*A.M.A. Arch. Ophthal.*, August, 1959) report on several conditions in which a low scleral rigidity seems especially common and in which it is necessary to be particularly wary in interpreting Schiotz readings. In the water provocative test it was found that the rise in intraocular pressure after water was greater in some eyes as determined by applanation than by Schiotz tonometry. Similarly, in endocrine exophthalmos a raised intraocular pressure was found in more eyes when measured by applanation tonometry. In myopia it was found that there was reduced scleral rigidity, and when using Schiotz tonometry there is a real possibility of underestimating intraocular pressure. Similarly, in glaucoma under treatment there is a reduction in scleral rigidity, and estimation of intraocular tension with the Schiotz tonometer will produce a low reading, whereas applanation tonometry reveals the true state of affairs.

Endothelial and Epithelial Dystrophy of the Cornea.

G. GORIN (*Amer. J. Ophthal.*, July, 1959) briefly discusses the pathology of corneal dystrophy and describes treatment. The author is of the opinion that the most important problem in the treatment of Fuch's dystrophy is to find a way of dehydrating the water-logged cornea. He has found that this is satisfactorily achieved with glycerine. The eye is anæsthetized, glycerine instilled, and the eye padded for 10 minutes. This is repeated daily for one week and then every second day. After treatment with glycerine, scopolamine is instilled and the eye covered with a pressure bandage. This treatment shortens the periods of recurrent attacks of hydration of the cornea, which in turn minimizes visual loss. It also tends to confine the disease to the central part of the cornea, an important consideration if grafting is contemplated. In mild cases patients can be kept comfortable and treatment repeated as soon as attacks of corneal hydration appear. The author reports on five cases.

OTO-RHINO-LARYNGOLOGY.

Prostheses in the Middle Ear.

W. HARRISON *et alii* (*A.M.A. Arch. Otolaryng.*, June, 1959) present a preliminary report on the use of prosthetic materials to reestablish continuity of the sound pressure transformer of the middle ear. The materials used were either fine polyethylene tubing (number 90, 50 or 20 according to circumstances) or gauge 40 tantalum wire. Several methods are described; these depend upon what part of the ossicular chain is remaining. In essence, there is created an artificial chain mechanism with one, two or three links by using prostheses to supply the missing elements. Polyethylene tube or tantalum wire can be attached to the long process of the incus and the other end sits on the footplate of the stapes. This reestablishes a three-ossicle chain. With absent incus, tantalum is attached to the handle of the malleus and the other end fashioned to fit on to the stapes footplate. Finally, when all ossicles are missing a columellar effect is obtained by placing a piece of tubing between the tympanic membrane and the stapedial footplate. Strict asepsis is essential and no infection or tissue reaction is reported. Gains in hearing in some cases have been strikingly good.

Osteomyelitis of the Frontal Bone.

H. P. SCHENCK (*Ann. Otol. (St Louis)*, June, 1959) reports on a series of 56 cases of osteomyelitis of the frontal bone associated with sinusitis, recorded between 1929 and 1958. This period is divided into three decades and the disease into that associated with subacute or chronic sinusitis and cases of primary fulminating type. In the first ten-year period, that is, before chemotherapy, the incidence of the fulminating type was nine out of 23 cases, with six deaths. In the middle period this was four out of 16 cases, with one death. In the third decade there were no fulminating cases and no deaths in 17 patients with associated subacute or chronic sinusitis. The author states that radiological evidence is very important. The use of chemotherapy delays the appearance of bone changes and the expected "moth eaten" appearance may not be obvious. Radiological examinations should be used to follow the course of the illness. Both immediate treatment and attention to any factor likely to lead to recurrence are important. The author suggests early drainage with an indwelling tube. Later, it is important to provide a permanent and free drainage pathway from the frontal sinus to the nasal cavity.

Primary Quinsy Tonsillectomy.

G. H. BATEMAN AND J. KODICEK (*Ann. Otol. (St Louis)*, June, 1959) report a series of 120 abscess tonsillectomies performed between December, 1949, and February, 1958. Endotracheal anæsthesia

is necessary, and it is noted that the use of relaxants can lead to obstruction of the airway during induction owing to œdema and loss of tone of the abductor muscles of the larynx which keep the airway patent. The patient should be in the head-low position. The operative technique is as usual, and post-operative care is as for interval tonsillectomy. Penicillin was given in all cases. No cases of septicæmia were reported and there were three secondary hæmorrhages, two being from the side opposite to the quinsy. No chest complications occurred. The authors point out that, as regards the site of the abscess, it was found that only 41% could have been incised with certainty; the remainder were either deep, posterior or inferior to the tonsil. In such cases the patient would have spent days of pain and trismus waiting for the abscess to point. This could be dangerous should the abscess point laterally into the parapharyngeal tissues or deep fascial planes of the neck. Abscess tonsillectomy provides a one-stage curative procedure. A final word of warning is given: that the operation should not be performed without the services of a skilled anæsthetist.

Adeno-Tonsillectomy and Deafness in Children.

B. J. McMAHON (*Laryngoscope* (*St Louis*), August, 1959) has investigated the incidence of poor hearing in 86 children who were undergoing adeno-tonsillectomy for reasons of health, and found that 45 had normal hearing, audiometrically and clinically, and 41 had poor hearing. Among these 41 children, hearing was restored to normal in 26, and not improved in 15, of whom four had nerve or mixed deafness. The important factor in the cases in which there was no improvement was probably attacks of otitis media, which occurred in 10 cases over a period of several years. The average age of patients at the time of operation was 7·8 years. The average duration of the history of poor hearing, with or without otitis media, was 2·4 years. He concludes that, judging by the definite improvement in hearing which followed adenoidectomy or adeno-tonsillectomy in children with poor hearing, with or without the complication of otitis media, it is advisable to perform this operation as soon as it is justified by the clinical conditions, regardless of the age of the patient. Procrastination may lead to irreparable damage to hearing. It should be the responsibility of oto-laryngologists to impress forcefully upon the general practitioner, the pædiatrician, the parents and the social service worker the significance of the earliest complaint by a child, or the earliest impression of the parent of difficulty in hearing.

Bell's Palsy.

H. L. WILLIAMS (*A.M.A. Arch. Otolaryng.*, October, 1959) discusses the problems facing the otologist in cases of Bell's palsy. The difficulty lies in the decision as to which patients should undergo nerve decompression, with the knowledge that delay until reaction of degeneration has occurred will usually result in incomplete recovery. On the other hand, many patients recover spontaneously, and these would have been subjected to an unnecessary operation, at which damage to the nerve is always possible. It appears that between 10% and 15% of patients do not recover spontaneously, but it is not possible to forecast with accuracy when reaction of degeneration will occur. The author quotes several other authors on this point without reaching a definite conclusion, and states that his practice is delayed operation in the knowledge that only 2% to 3% of patients have serious disfigurement if this course is followed. He then discusses the problem of when to operate on the small percentage in whom recovery does not become evident and, as a working rule, he states that if no sign of beginning regeneration is indicated by electromyography after two months, the facial nerve should be decompressed.

Mobilization—A Long-Term Report.

S. ROSEN AND M. BERGMAN (*Laryngoscope* (*St Louis*), August, 1959) present a follow-up report on the first 100 cases of hearing improvement by stapes mobilization. Seven years after the first successful operation, the authors have endeavoured to evaluate the results by finding which patients have retained maximum improvement, which have regressed but still retain good improvement over their pre-operative level of hearing, which have regressed to their pre-operative level, and how many of these have had revision operations. They state that of the 100 patients, 83 have maintained significantly improved hearing for periods of from three to seven years.

Oval Window and Round Window Surgery in Extensive Otosclerosis.

W. F. HOUSE (*Laryngoscope* (*St Louis*), June, 1959) presents a preliminary report of a new technique in cases where the otosclerotic focus is so extensive as to make the routine methods impossible. A small diamond burr is used to thin down the footplate. The head and crura of the stapes having been removed, a polyethylene prosthesis is placed between the lenticular process of the incus and the footplate. The author also reports some cases where the round window is involved and gives details of some cases where the diamond burr is used to remove this otosclerotic focus. He urges caution with the patients who are not severely deaf, as in their cases round window surgery can be detrimental.

Endocrine Aspects of Ménière's Disease.

G. E. SHAMBAUGH (*Laryngoscope* (*St Louis*), August, 1959) briefly explains the enzymatic concept of allergy and how, with insufficiency of thyroid hormone within the cells, there is a reduction in the formation of normal type proteolytic enzymes. Instead, abnormal enzymes are produced which only partially proteolyse foreign proteins (including the allergens) with production of highly toxic compounds, which include histamine. The relationship between endolymphatic hydrops and allergy has been noted by many authors. The author states that in 28% of a small series of cases of hydrops that failed to respond to dilute histamine, correction of a deficiency of thyroid metabolism resulted in a greater relief of symptoms than would probably have occurred in the normal fluctuations in these cases. The usual deficiency was the condition known as the hypometabolic syndrome with normal protein-bound iodine level, but lowered basal metabolic rate when this was estimated after sedation. In this condition desiccated thyroid is not effective, whereas triiodothryonine can relieve the hypometabolic symptoms.

Repair of Ossicular Defects with "Ostamer".

J. SATALOFF (*A.M.A. Arch. Otolaryng.*, October, 1959) has used "Ostamer", a polyurethane foam, to repair defects in the ossicular chain in two live subjects. In three years there has been no tissue reaction in the first human implant. "Ostamer" is a mixture of a prepolymer with a catalyst, the mixing being done in the operating theatre at the time of use. There are certain difficulties in the use of this substance, one of which is the speed of its hardening, which allows only very short time to place it and make adjustments. Also, it swells on hardening, and it is difficult to assess the amount to use, thus there is an excess of the "Ostamer". In the two patients there was substantial gain in hearing.

SURGERY.

Arteritis after Correction of Coarctation of the Aorta.

A. O. SINGLETON, JR., L. M. S. McGINNIS AND H. R. EASON (*Surgery*, April, 1959) state that there have been previous reports of arterial inflammation, necrosis and thrombosis of smaller distal vessels after the correction of coarctation of the aorta. This has been limited to vessels below the coarctation and has developed in the immediate post-operative period. They state that only five cases of such a condition have been reported previously in the literature. Two additional cases are reported, one of which did not develop for three months after the operation for coarctation. Although involvement of many vessels has been demonstrated by the pathologist, in previously reported cases the clinical picture has been that of mesenteric thrombosis. However, in one of the authors' cases evidence of ischæmia of the spinal cord was also present. The authors point out that many surgeons have reported their experiences with coarctation of the aorta, and that the main complications after resection have usually been due to technical operative difficulties such as dehiscence of the aortic suture line, or to preexisting cardio-vascular disease such as cerebral hæmorrhage and cardiac failure. However, acute necrotizing arteritis of the abdominal organs developing in the post-operative period is becoming more frequently reported. Autopsy reveals inflammation of the small arteries and arterioles confined to the body area supplied by the aorta below the coarctation, often with numerous infarcts in the liver, spleen and intestine. Clinically, these patients present with mesenteric thrombosis, often requiring bowel resection. The authors consider these lesions to be primarily the results of changes in circulatory dynamics after operation.

British Medical Association.

VICTORIAN BRANCH: SCIENTIFIC.

A MEETING of the Victorian Branch of the British Medical Association was held on October 15, 1958, at Prince Henry's Hospital, Melbourne. The meeting took the form of a series of clinical demonstrations by members of the honorary medical staff of the hospital.

Tænia Saginata Infestation Treated with "Atebrin".

DR. T. J. F. FRANK showed a male patient, aged 30 years, a carpenter, born in Lebanon and resident in Australia for 11 years, who had been first examined on April 2, 1958, when he said that he had been passing segments of worms for the last eight years. Apart from occasional abdominal pain, he had enjoyed good health. Whilst in Lebanon he had frequently eaten raw and incompletely cooked beef. He was a healthy young man, and showed no abnormality on routine medical examination. Macroscopic examination of the fæces on April 24 revealed eight tapeworm segments, whilst on microscopic examination numerous tapeworm ova were seen. The size of the proglottides suggested a *Tænia saginata* infestation.

The following treatment, as suggested by D. F. Sandars (MED. J. AUST., May 3, 1958) was ordered. On June 1 and 2, a light diet was taken; on June 3, complete starvation was observed, only black coffee or tea with sugar and no milk *plus* glucose drinks being permitted. At 8 p.m., one ounce of Glauber's salts dissolved in a glass of water was given. Next morning at 9 o'clock the patient was given ten 0·1 gramme tablets of "Atebrin Hydrochloride" in a glass of water containing one teaspoonful of sodium bicarbonate. At 11 a.m., one ounce of Glauber's salts was given. Several hours later there were frequent motions; in the first, numerous ripe proglottides, then short lengths of the worm and immature proglottides were passed. Finally, the scolex with portion of the worm was passed. Laboratory examination showed that the tapeworm was *Tænia saginata*. When the patient was examined on July 4, no more segments had been passed. The only disability from treatment was loss of libido, which returned to normal within one month. When he was seen on October 7, he felt well and his fæces were normal.

Dr. Frank commented that the treatment was simple to give and most effective. Although *Tænia saginata* infestations were endemic in Australia, few cases were encountered. However, with increasing numbers of immigrants it was probable that such infestations would be seen more frequently, and also other tapeworm infestations.

Coronary Sclerosis, Hiatus Hernia, Calcified Thyroid Adenoma.

Dr. Frank next showed a male clerk, aged 52 years, who had been examined on April 18, 1958, with a history of dyspnœa, and of aching pain over the heart and in the left epigastric area for the past fifteen months on going up hills; it was relieved by resting and lying down. Pain also occurred over the heart when he was worried. For years he had noted flatulent dyspepsia and acid eructations, particularly on stooping. Fifteen months earlier he had had an attack of heart pain, and was rested for three months. An electrocardiogram in May, 1957, suggested coronary disease. He had had three attacks of pneumonia, and for many years he had had many worries, mainly because of his wife's ill-health and the loss of a child, aged 12 years, from hydrocephalus and pneumonia.

Examination of the patient showed him to be short and plump; he weighed 11 st. 13 lb. There was a firm nodule in the left lobe of the thyroid. The heart was slightly enlarged, the aortic second sound was markedly accentuated and his blood pressure was 180/120 mm. of mercury. Pruritus ani and piles were also present. An X-ray examination of the thyroid region showed a calcified adenoma, approximately 5 cm. in diameter, in the left lobe of the thyroid. An X-ray film of the chest revealed an increase in the transverse diameter of the heart. X-ray examination with a barium bolus revealed a small diverticulum on the left anterior wall of the œsophagus just below the level of the aortic arch. A barium meal X-ray examination revealed a sliding hernia of the gastric fundus with free reflux into the lower end of the œsophagus. A resting electrocardiogram was normal, but after effort there was slight but delayed and persistent S-T depression in lead V, whilst the *T* wave in lead V_5 was biphasic, the changes suggesting myocardial ischæmia. As a result a diagnosis of coronary sclerosis with myocardial ischæmia, non-toxic thyroid adenoma, hiatus hernia, œsophageal diverticulum, overweight and nervous exhaustion was made.

Dr. Frank remarked that the occurrence of hiatus hernia and coronary sclerosis was well known, and in the case under discussion each had classical symptoms. Probably repair of the hiatus hernia would have relieved the patient's dyspeptic symptoms. As the thyroid adenoma, apart from a swelling, was asymptomatic, no treatment was advised. In his treatment weight reduction and the necessity for sensible living were stressed; sedation was ordered to relieve his nervous state.

Left Nephrectomy for Hypertension.

The third patient shown by Dr. Frank was a carpenter, aged 45 years, who had been examined on August 18, 1958, when he gave the following history. For several years he had suffered from mild hypertension, and in April, 1957, he had developed left Bell's palsy, from which an incomplete recovery had been made. For the past three months he had noticed shortness of breath and retrosternal pain on effort. In recent weeks, he had also had difficulty in expressing himself, had been giddy, and had suffered from several headaches, which were rapidly relieved with A.P.C. mixture. He had passed urine several times at night, but had had no difficulty and no dysuria. Five years earlier he had had hæmaturia, and had subsequently passed a stone. Full investigations including intravenous pyelography and cystoscopy, gave normal results. There was no history of scarlet fever, diphtheria or nephritis. His father had died at the age of 76 years from an unknown cause; his mother had died at the age of 30 years from a kidney fit at childbirth. Two brothers and one sister were alive and well.

Examination showed the patient to be a well-built man, with some pallor and residual left Bell's palsy. He had difficulty in expressing himself clearly. The heart was enlarged, and loud mitral and aortic systolic murmurs were present. The blood pressure was 260/130 mm. of mercury. The urine was "half solid" with albumin, but no sugar was present. A microscopic examination of the urine revealed a few red cells and an occasional leucocyte, but no casts. Examination of the optic fundi showed considerable retinopathy of a rather unusual appearance. The arteries showed variations in calibre with regions of extreme attenuation, typical of the changes of malignant hypertension; there was also some sclerosis of the arteries as shown by the crossing changes. Punctate hard exudates were numerous between the macula and the disk. Numerous deeper and a few superficial hæmorrhages were noted. The blood urea content was 47 mm. per 100 ml., the urea concentration was 1·80% at the end of two hours, the urea clearance was 62% of normal and the serum protein content was 7·1 grammes per 100 ml. An X-ray examination of the chest revealed a great increase in the transverse diameter of the heart, a prominent aortic notch and increased density of the aorta. Intravenous pyelography revealed a normal right kidney and an almost non-functioning left kidney, which was probably much smaller than the right. A cystoscopic examination gave normal results; retrograde pyelography revealed no ureteric obstruction.

On August 29, 1958, the left kidney was removed. Examination of the kidney revealed it to be small and nodular, with many cysts on the surface. The ureter was dilated and there was some pelvic hydronephrosis. The renal tissue was much reduced. Microscopic examination revealed marked loss of glomeruli, slight thickening of the endothelium of the smaller arteries, patchy infiltration with lymphocytes, fibrosis and œdema. The kidney was suggestive of pyelonephritis associated with essential hypertension rather than of true pyelonephritis due to obstruction. On October 2 the patient felt much improved; his blood pressure was 210/130 mm. of mercury, and his urine contained only a faint trace of albumin.

Dr. Frank said that, according to Homer Smith, unilateral nephrectomy was helpful to 2% of all hypertensive patients. The slow fall in blood pressure since nephrectomy made him guarded in his prognosis. In the recorded literature the best results after nephrectomy for hypertension had been achieved in younger patients with an atrophic kidney; 50% of patients in one such series of cases had a permanent reduction of blood pressure. Even in obvious unilateral disease, nephrectomy was an experimental procedure and subject to error.

Aneurysm of the Ascending Aorta.

Dr. Frank finally showed a storeman, aged 51 years, who had been examined on March 22, 1957, when he gave the following history. He had been treated for fibrositis of

the upper right portion of the chest wall, the back of the right side of the chest and the right shoulder joint during the winter of 1956 by injections, physiotherapy and "Butazolidin" tablets. An X-ray examination of the chest at that time had given normal findings. Only slight improvement had occurred. Pain was still present in the same region, and was aggravated by exertion, coughing, stooping and sneezing. His past history was clear, and he denied a history of venereal disease. He had two children, aged 24 and 19 years, and four brothers alive and well. One brother, aged 56 years, had died of peptic ulcer and alcoholism.

On examination, the patient was found to be a healthy man, with tenderness in the intercostal muscles of the right upper aspect of the chest wall and of the right interscapular region. The heart size and sounds were normal. His blood pressure was 140/80 mm. of mercury. The central nervous system was normal. An X-ray examination of the chest on March 25, 1957, was reported on in the following terms:

> Oval homogenous soft tissue mass is present in the superior mediastinum anteriorly, approximately four inches in length and three inches across. It has fairly well defined borders and extends slightly to the right where it projects somewhat into the right lung. Posteriorly, there is some displacement of the trachea backwards to the right.

On fluoroscopic examination the mass failed to show any pulsation and appeared to lie close to the aortic arch, but was not continuous with it. The heart and great vessels showed no abnormality apart from some displacement of the aortic knob to the left. The lung fields were clear. An X-ray examination of the thoracic part of the spine revealed no abnormality apart from very slight spondylitis of the mid-thoracic section. It was concluded that the lesion was an upper mediastinal tumour or cyst, possibly a dermoid, thymic or tracheo-bronchial cyst. Examination by a nose and throat specialist revealed a normal larynx and chronic tonsillitis.

On March 29 the patient was referred to a surgeon, who considered that he had a mediastinal cyst, possibly hydatid. On April 11, thoracotomy revealed a saccular aneurysm of the ascending aorta just above the aortic valves. A nylon blanket was applied. The patient's voice became husky after operation, and the huskiness had persisted. His blood reacted strongly to the Wassermann test, fixing 24 M.H.D. of complement. Treatment had consisted of several courses of bismuth injections with mercury and iodides given orally. No penicillin was given, as the patient had a marked post-operative allergic reaction to that antibiotic. An X-ray examination of the chest on July 12 revealed no increase in the size of the aneurysm, and the lung fields were clear. He still complained of a peculiar pain in the right upper quadrant of his chest, but much less marked than previously. Giddiness had persisted, his heart sounds were normal and he was able to work efficiently at his occupation.

Dr. Frank commented that in the case under discussion a cystic mass in the upper mediastinum was correctly diagnosed only at operation and shown to be an aneurysm. As a result of operation, it had not increased in size. Anti-specific treatment so far had not produced any effect on the Wassermann reaction. The huskiness of the voice was apparently secondary to the nylon blanket applied around the aneurysm.

Pneumonia With Unusual Features.

DR. R. GAULD showed four patients, to demonstrate unusual features of pneumonia at the hospital over the past year.

The first patient, a man, aged 39 years, had suddenly become ill 24 hours before his admission to hospital. He complained of pleuritic pain in the right upper quadrant of the chest, malaise and shortness of breath, and coughed up rusty sputum. He was given 1,500,000 units of penicillin, one gramme of streptomycin and three grammes of "Sulphatriad" by his local doctor. On his admission to hospital, his temperature was 102·4° F., his respirations numbered 36 per minute and his pulse rate was 152 per minute. There were signs of consolidation of the upper lobe of the right lung, and a friction rub was present. The leucocytes numbered 15,000 per cubic millimetre. The history and findings were typical of pneumococcal lobar pneumonia; however, he failed to respond to four days of treatment with penicillin, 2,000,000 units each day, and sulphadimidine. Sputum cultures produced a slight growth of *Staphylococcus aureus*, resistent to penicillin and sulphonamides. The patient was therefore given achromycin. The response was dramatic, and he had remained well ever since.

The second patient shown by Dr. Gauld was a male textile worker, aged 50 years, who had become ill eight weeks before his admission to hospital. He complained of a sudden onset of malaise, fever, cough with a small amount of yellow sputum and pleuritic pain on the right side. Two weeks later he coughed up a large amount of blood-stained yellow sputum. He continued to cough up large amounts of yellow sputum and lost over a stone in weight. On his admission to hospital he was cachectic, his temperature was 101° F., his fingers were clubbed, there were rales over the right upper quadrant of the chest, and a friction rub was heard. An X-ray examination of his chest revealed gross consolidation and multi-loculated cavities throughout the upper lobe of the right lung. Sputum examination revealed normal flora and no tubercle bacilli. The Mantoux response to old tuberculin, 1: 100, was negative. Bronchoscopic examination revealed no bronchial abnormality. Treatment consisted of the administration of penicillin, 750,000 units every six hours for seven weeks, and "Achromycin", 750 mg. every six hours for 17 days. At the end of this period, although there had been marked improvement in the radiological signs, cavitation was still present. Resection of the upper lobe of the right lung was considered, but because of his lack of symptoms and the continued improvement in the X-ray findings, this was not carried out. He had since remained well, and an X-ray film taken eight months later showed considerable distortion of the upper lobe of the right lung, apparently due to fibrosis, and no obvious cavities. He was considered to have had chronic suppurative pneumonia.

Dr. Gauld then showed a male patient, a gardener, aged 53 years, who had had attacks of bronchial asthma for 30 years. Two weeks before his admission to hospital he had developed a large carbuncle in the lumbar region. One week later he developed pneumonia, and he had been given an unknown amount of penicillin and tetracycline by his local doctor. On his admission to hospital he was ill, with mental confusion, and shortness of breath, and was coughing up large amounts of blood-stained sputum. The carbuncle had practically healed. On examination of the patient, his temperature was normal, and there were signs of emphysema and scattered rales and rhonchi over both lungs. Friction rubs were present over the lower lobes of both lungs. An X-ray examination of his chest showed consolidation, with a large cavity in the upper lobe of the right lung, and there were five thick-walled abscess cavities about one inch in diameter in the left lung. Sputum cultures grew normal flora only and no tubercle bacilli. The leucocytes numbered 15,000 per cubic millimetre. He was given penicillin, 1,000,000 units three times a day, for five weeks, and erythromycin, 500 mgm. every six hours for eight days. At the end of that period he had no symptoms and the X-ray findings were practically normal. Dr. Gauld said that in spite of the fact that there were no staphylococci in the patient's sputum, that organism was considered to be the cause of his multiple lung abscesses and pneumonia. Presumably the organism had reached the lungs by the blood-stream from the carbuncle.

The last patient shown by Dr. Gauld was a carpenter, aged 52 years. Eighteen months before his admission to hospital he had been treated for pneumonia and an X-ray examination had revealed partial collapse and consolidation of the middle lobe of the right lung. He was well, with slight cough and sputum, until six weeks before his admission to hospital, when he noticed increasing weakness, cough with blood-stained yellow sputum, right pleuritic pain and loss of weight. On examination of the patient, his fingers were clubbed and there were signs of collapse of the lower lobe of the right lung. An X-ray examination revealed consolidation of the apical and subapical segments of the lower lobe. Sputum cultures revealed no specific organism, and no cancer cells were seen. Bronchoscopic examination revealed a flat piece of bone about half an inch square embedded in granulation tissue in the right lower lobe bronchus, just below the middle lobe orifice. That foreign body was removed, and the patient made an uneventful recovery. A bronchographic examination made some weeks later revealed no abnormality.

Pulmonary Embolism.

DR. H. IAN JONES presented four cases of pulmonary embolism. The diagnosis was definite in three and probable in one. In none did a recent operation, prolonged recumbency or heart disease precede the pulmonary embolism. In three there was no clinical evidence of venous thrombosis in the limbs or elsewhere immediately preceding the pulmonary episodes.

The first patient discussed was an estate agent, aged 57 years, who two years previous to his admission to hospital had been treated for hypertension (blood pressure 210/110 mm. of mercury) and obesity. In November, 1954, one year later, he was submitted to appendicectomy, his convalescence being uneventful. In particular there were no thrombotic complications. Thirteen months later, on December 12, 1955, he noticed pain in the right side of his chest while gardening. The pain, which was pleuritic in character, rapidly became worse and was associated with fever, a dry cough and signs of consolidation in the middle lobe of the right lung. A diagnosis of pneumonia was made and he was treated with antibiotics, but over the next week his condition became progressively worse. He was dyspnœic and cyanosed, and developed gallop rhythm with falling systemic arterial blood pressure and rising jugular venous pressure. Signs of pleural effusion developed at both lung bases, and he had attacks of pain across the front of the chest simulating angina. There was no clinical evidence of venous thrombosis in either leg. There was no hæmoptysis, and the little sputum he produced yielded normal flora. The leucocyte count rose to 20,000 cells per cubic millimetre, and a neutrophil leucocytosis was present. The electrocardiogram showed a prominent *S* wave and depression of the *S-T* segment in lead I, depression of the *S-T* segment and a "staircase" effect in lead II, a small *Q* wave in lead III and flat inversion of the *T* wave in lead III and right ventricular V leads.

The significance of the electrocardiographic changes was not recognized at the time, and it was thought that the diagnosis of pneumonia with associated myocarditis was most probable. Chest aspiration failed to produce more than one or two millilitres of blood-stained fluid on two occasions. The patient died suddenly on January 3, 1956. Autopsy revealed thrombosis of the left external iliac vein, extending along the common iliac vein to the inferior vena cava. Scattered throughout both lungs were multiple pulmonary infarcts of varying age and size. The largest in the lower lobe of the right lung measured 5 cm. square. There was no evidence of myocardial infarction. Microscopic examination of both kidneys revealed changes of pyelonephritis.

The second case discussed was that of a sheep farmer, aged 35 years, who 17 years previously had had an attack of measles complicated by a deep venous thrombosis in the left leg. The leg had caused him no trouble since, and he had remained a particularly active man. While watching a football match on August 2, 1958, he was suddenly stricken with pain in the left shoulder and lower down on the left side of the chest. The pain was pleuritic from the start, and severe enough to cause considerable distress. Later that night he coughed up a little bright blood, and he continued to do so during the next four or five days. When he was examined on the day after onset, he showed signs of a left pleural effusion and elevation of the left hemidiaphragm. There was no pain or tenderness in the left leg, and Homan's sign was absent. X-ray examination of the chest on August 4, confirmed the presence of a pleural effusion with considerable elevation of the left hemidiaphragm. The patient was febrile, and producing a little blood-stained sputum which on culture produced normal flora. The leucocytes numbered 15,000 cells per cubic millimetre, and neutrophilia was present. Anticoagulant therapy was commenced on August 4 (heparin for 36 hours and "Dindevan"), and on August 7 he developed pain, tenderness and swelling in the left calf, and Homan's sign was present. With continuation of "Dindevan" therapy, the leg symptoms disappeared in a few days, he became afebrile, and the cough, sputum and chest pain disappeared. A chest X-ray examination on August 18, revealed persisting elevation of the diaphragm and shadowing in the posterior basal region of the left lung due to pleural thickening and an infarcted lung. The effusion was not aspirated. The administration of anticoagulants was continued for six weeks, and as far as was known the patient had remained well since.

The third case discussed by Dr. Jones was that of a fitter, aged 36 years, who had been in good health until three weeks before his admission to hospital, when he developed an upper respiratory tract infection. One week later he developed right-sided pleuritic pain lasting three or four days, with associated malaise and night sweats. He had a dry cough, which was with few exceptions unproductive throughout the illness. On one or two occasions he produced a slight amount of brownish sputum. He was treated with penicillin and subsequently tetracycline before his admission to hospital, presumably owing to persistence of signs in his chest.

Almost two weeks after the onset of right-sided pleurisy, he was suddenly afflicted with pleural pain on the left side, accompanied by considerable dyspnœa and distress. He was admitted to hospital on the following day, September 20, 1958, still in pain, dyspnœic and pale and with an unproductive cough. There was no hæmoptysis, and at no time did he show any clinical evidence of venous thrombosis in the limbs. There were physical signs of pleural effusion and consolidation on both sides posteriorly. A chest X-ray examination revealed bilateral elevation of the diaphragm, with a large pleural effusion on the right and a smaller one developing on the left. The heart was clinically normal and the electrocardiogram was normal. Aspiration of the right pleural effusion and subsequently the left yielded evenly blood-stained sterile fluid. Culture of the sputum produced normal flora only, and there was no leucocytosis. He was treated with penicillin, but his condition deteriorated over the first week in hospital. An exacerbation of the left-sided pain was associated with an increase in effusion, tightness across the chest and increasing fever. A further E.C.G. showed some elevation of the *S-T* segment in leads I and II, and AVL only. Anticoagulant therapy ("Dindevan") was instituted, and the antibiotic "cover" was widened to include streptomycin. There were no further episodes, the temperature gradually returned to normal over two weeks and the chest gradually cleared. Marked subjective improvement was noticeable within a few days of commencement of anticoagulant treatment. Other investigations included blood cultures, a Mantoux test (1:1000 old tuberculin), and examination of the sputum for tubercle bacilli and of the blood for cold agglutinins—all with negative results. He now had no physical signs in the chest, but the latest X-ray film on October 10, still showed some consolidation in the base of the lower lobe of the right lung, and a very small effusion in the left costophrenic angle. He was still receiving anticoagulant therapy and taking penicillin tablets by mouth, although it was thought that the latter could be stopped.

Dr. Jones said that although the diagnosis of pulmonary infarction was not proven, six points taken together were strongly in its favour: (i) the occurrence of bilateral pleurisy affecting first one side and then the other; (ii) the fact that both effusions were blood-stained; (iii) the virtually unproductive cough throughout the illness; (iv) bilateral elevation of the diaphragm; (v) absence of response to a wide range of antibiotics; (vi) consistent improvement dating from the commencement of anticoagulant therapy, which had been maintained.

Dr. Jones finally presented the case of a housewife, aged 42 years, who 13 years previously had suffered a pulmonary embolus after a pelvic operation. Since then she had suffered from occasional episodes of pain in the calves of her legs with recurrent attacks of sharp pain in the chest. Six days before her admission to hospital on October 5, 1958, she had noticed pain in the left calf, followed by tightness, swelling and tenderness two days later. Three days later pleuritic pain developed in the left side of the chest, becoming more severe and associated with dyspnœa. There was a dry cough, but no hæmoptysis. On her admission to hospital, the calf of the left leg was found to be tense and tender, Homan's sign was present, and there were signs of consolidation in the lower lobe of the left lung posteriorly. An X-ray examination of the chest confirmed the presence of an infarct in that region. The electrocardiogram was normal, and there was no leucocytosis. The patient was febrile. With anticoagulant therapy the temperature gradually subsided over nine days and the signs in her chest cleared rapidly, but she still had Homan's sign and some tenderness of the calf. In addition to the thrombo-embolic disorder, the patient showed an hysterical contracture of the fingers of the right hand, involving mainly the index and middle fingers, associated with complete hemianæsthesia involving the whole of the right side of the body. The deformity and hemianæsthesia dated from a trivial injury to the dorsum of the hand three years earlier, when the patient was under considerable emotional stress.

Dr. Jones said that the cases he had presented illustrated the following points, which often led to difficulties in the diagnosis of pulmonary embolism. (i) Spontaneous venous thrombosis occasionally occurred in otherwise healthy, active adults. (ii) Venous thrombosis in the limbs might occur in the absence of clinical evidence of its presence. (iii) Pulmonary emboli might then occur as the presenting feature of the illness. Clinical evidence of venous thrombosis in a limb might appear some days later, or might not appear at all. (iv) The leucocyte count might vary from normal to high in the absence of infection. (v) Hæmoptysis was frequently absent in non-cardiac cases of pulmonary embolism. (vi) Infarcts did not usually produce wedge-shaped shadows in a chest X-ray film. They were

more often rounded or indefinite in outline, and basal infarcts were frequently obscured by pleural effusion and elevation of the diaphragm. Failure to recognize the syndrome might well result in a fatal termination.

Chronic Membranous Nephritis.

DR. M. G. WHITESIDE presented a man, aged 67 years, who had given a two years' history of severe peripheral œdema, and who was found to have gross albuminuria. The diagnosis of nephrotic syndrome was supported by the finding of high cholesterol and very low albumin levels in the serum. Renal function tests indicated only partial impairment. Intravenous pyelography revealed a non-functioning right kidney, and because of that and the patient's age, an attempt was made to demonstrate a unilateral renal lesion as the cause of the syndrome. However, retrograde pyelographic findings were normal, catheter specimens from each ureter contained approximately equal amounts of albumin, and venography of the inferior vena cava showed a patent right renal vein. Finally, needle biopsy of the kidney (performed by the Walter and Eliza Hall Institute, Royal Melbourne Hospital) gave the diagnosis of chronic membranous nephritis. Response to steroid treatment was unsatisfactory.

Dr. Whiteside showed histological sections from renal biopsy material in other cases of the nephrotic syndrome, to illustrate the diversity of causes of that condition and the value of renal biopsy in its diagnosis.

Laboratory Aids to the Diagnosis of Myocardial Infarction.

DR. ALLAN WYNN said that a study of the diagnostic value of estimation of the serum glutamic oxalacetic transaminase (S.G.O.T.) level in patients with acute myocardial infarction had been made in conjunction with Miss Anne Carmichael, of the Biochemistry Department, and Dr. H. Dodge, Medical Registrar. The "Sigma" colorimetric method of estimation was used throughout, and was found to be extremely satisfactory. The results were reproducible, and compared well with those obtained simultaneously by the spectrophotometric method ("Karmen"). In normal patients without liver disease the S.G.O.T. level was usually below 25 units; in patients with left ventricular failure, congestive cardiac failure or repeated attacks of angina pectoris, the S.G.O.T. level was rarely above 40 units. However, in severe congestive cardiac failure with advanced congestion of the liver, an abnormal increase in the S.G.O.T. level might occur. One important technical consideration in performing that test was that every precaution had to be taken to prevent hæmolysis from occurring in the sample because of the transaminase liberated from hæmolysed red cells.

Dr. Wynn went on to say that in 30 cases of acute myocardial infarction, the transaminase level was abnormal in 28, equivocal in one and normal in one. Estimations should be performed daily until a stable level was reached. It was usually found that the highest level occurred in the first 48 hours after the attack, and that a normal level was reached within five days. In the series under discussion there were four cases in which the electrocardiographic changes were only slight, two in which the electrocardiogram at first was normal and became abnormal within a week, and two in which the electrocardiogram remained unchanged, although there were extensive abnormalities due to previous myocardial infarction. The erythrocyte sedimentation reached a level greater than 25 mm. in the first hour during the first week after the attacks in 19 cases. In the series eight patients died, and recent myocardial infarction was demonstrated *post mortem*. In all those patients, the S.G.O.T. levels were very high. It was found that there was a definite correlation between the height of the S.G.O.T. level and the extent of myocardial infarction; but some patients who seemed clinically to have had small infarcts also had high S.G.O.T. levels.

Dr. Wynn commented that it was probable that the main value of the estimation would be for patients who seemed clinically to have myocardial infarcts, and whose electrocardiogram was normal at first or else showed no change from a previous abnormality. A study to determine the value of the test in the differential diagnosis of acute myocardial infarction from pulmonary embolism was proceeding.

Bone Metastases from Malignant Disease.

DR. G. NEWMAN-MORRIS presented cases and X-ray films of bone metastases from various forms of malignant disease, and made the following points: (i) In 25% of cases of malignant disease, secondary deposits in bone were present, and almost invariably the metastases were multiple. (ii) Metastases appeared most commonly from malignant disease of the prostate, breast, kidney and stomach. (iii) Common sites were the spine, pelvis, ribs, skull, femur and humerus.

In the first case discussed by Dr. Newman-Morris, the main clinical feature was a silent primary tumour. The patient was a man, who had an insidious onset, fairly brief, of a swelling in the medial condyle of the knee. Post-mortem examination finally revealed malignant disease in the pancreas and adrenals.

The second case was that of a girl, aged 27 years, who had been admitted to the hospital with the diagnosis of acute rheumatoid arthritis, because of multiple pains involving mainly the lumbar part of the spine. A small tumour in the breast was found shortly after her admission, and a breast biopsy and bilateral oophorectomy were carried out shortly afterwards and a bilateral adrenalectomy a fortnight after the first operation. The patient, while in hospital, suffered a pathological fracture of the sternum and died from respiratory embarrassment. X-ray films showed possible radiological changes in the pelvis and both shoulders.

The next case was one of pathological fracture. The patient was a woman, aged 67 years, who five years before had had a mastectomy for a tumour in the breast, but there was no evidence of glandular involvement. The patient had recently fallen and fractured the neck of her femur, and X-ray examination revealed an osteolytic metastatic carcinoma. She had refused adrenalectomy.

The fourth case was that of a man who had undergone nephrectomy a year before, and at that time a malignant embolus was seen in the renal vein. He had been admitted to hospital with pathological fractures in the forearm and femur.

Dr. Newman-Morris finally showed X-ray films to demonstrate osteoplastic secondary deposits from prostatic carcinoma.

Carcinoma of the Kidney.

MR. D. DONALD presented a case of carcinoma of the kidney which had presented as epididymo-orchitis; on investigation, the patient was found to have hydroureter and hydronephrosis. Carcinoma of the kidney was found at operation. Mr. Donald wondered whether the other conditions provided the ætiology.

Cholelithiasis in a Child.

Mr. Donald also presented the case of a girl, aged 12 years, suffering from cholelithiasis. He said that the condition had been undiagnosed at eight previous consultations. There were metabolic considerations in the ætiology. X-ray examination of the bladder revealed 18 stones, yet only one was found at operation. Mr. Donald discussed the management, and condemned the placing of tubes in the common bile duct, except in rare circumstances.

Pancreatico-Duodenectomy for Carcinoma of the Ampulla of Vater.

MR. W. J. MCCANN showed a patient who had had an eleven months' history of obstructive jaundice. After pre-operative preparation with vitamin K injections, ascorbic acid, blood transfusion, and a diet rich in protein and carbohydrate, laparotomy revealed a grossly distended gall-bladder, and a lump the size of a thumb nail at the ampulla of Vater. No liver or lymph-node metastasis was seen, and a one stage pancreatico-duodenectomy was performed. Reconstruction consisted of the anastomosis of a jejunal loop apex to the bile duct, and then anastomosis of the afferent limb to the pancreatic duct and pyloric antrum. Below that, an entero-anastomosis was performed to help the gastric contents to bypass the biliary and pancreatic anastomoses.

Mr. McCann pointed out that the five-year survival rate of that operation for ampullary neoplasm was 38·4% at the Mayo Clinic and 30% at the Lahey Clinic—a much better prognosis than that which could be given for neoplasms of the stomach, lung, œsophagus, etc.

Biliary Dyskinesia.

Mr. McCann then discussed the surgical physiology of biliary dyskinesia due to spasm of the sphincter of Oddi, and explained the morphine cholangiogram test for biliary dyskinesia.

Patients were also shown both before and after biliary and pancreatic sphincterotomy. The patients had presented originally with the diagnosis of post-cholecystectomy syndrome.

Thrombosis in Cerebral Vessels.

DR. ARTHUR SCHWIEGER presented a demonstration setting out the anatomical basis and clinical syndromes in occlusion of the cerebral arteries, illustrated by appropriate sketches and arteriograms. Two patients were shown as examples of classical internal carotid artery thrombosis and thrombosis of the middle cerebral artery. In discussing treatment, Dr. Schwieger considered the use of anticoagulant therapy.

Urinary Tract Infection.

MR. A. B. ALDER presented a demonstration designed to emphasize the necessity for urological investigation of patients presenting with urinary tract infection. A broad outline of the anatomical sites and pathological causes of infection was presented, together with the management in individual cases. Mr. Alder discussed several patients who had been treated for some months with various antibiotics with temporary improvement. The first, a man, aged 64 years, required a cystectomy with ureteric transplantation for a carcinoma of the bladder. The second, a girl, aged 10 years, underwent a Scardini-Culp pyeloplasty for a pelvi-ureteric junction stricture, with subsequent clinical cure. The third, an unmarried woman, aged 24 years, responded dramatically to nephro-ureterectomy for pyonephrosis and hydroureter due to tuberculosis with secondary infection. Among other conditions illustrated were bladder-neck obstruction, cervicitis and urethritis in the female, and chronic pyelonephritis.

Mr. Alder pointed out that the management of an acute infection was with a selected drug until such time as investigation was warranted.

Dermatological Demonstration.

DR. ERIC H. TAFT illustrated dermatological problems with coloured transparencies and demonstrated many of the commoner dermatoses and tumours seen in practice. In particular, great emphasis was placed on the skin as the largest organ in the body, and also as a labile organ whose response to friction, temperature change, secondary infection and unsuitable local treatment often obscured the original condition. Those complications were illustrated by changes produced in contact dermatitis. Numerous examples were shown of areas of dermatitis which had initially been produced by contact factors, but which had been converted by friction and unsuitable local treatment into persistent areas of neurodermatitis. Many of them had been present for months after the original contact, which was often trivial in nature, had caused the patient great distress and had been a source of difficulty to the attendant practitioner, the patient's employer and, occasionally, the insurance company in cases which were accepted as occupational.

Psychosomatic influences in dermatology were illustrated in neurodermatitis, trichotillomania, neurotic excoriations and dermatitis artefacta.

The differentiation between various types of tumours was discussed, some of the commoner metabolic skin diseases were mentioned, the dermatological manifestations of diabetes, hyperlipæmia and internal malignant disease were detailed, and the morphological features of fungous infection of the skin were shown and its management was discussed.

Diabetes.

DR. J. R. STAWELL and DR. H. BREIDAHL demonstrated and talked on the pitfalls in the management of diabetes. They discussed the following points.

Diets.

It was pointed out that the prescription of the correct diet when a patient was first examined was of paramount importance. Case histories were shown illustrating the common faults, particular stress being laid on the necessity of giving adequate food in conjunction with insulin to the underweight, and the use of a low-calorie diet for the overweight, when insulin was seldom necessary.

Syringes.

The various types of syringes available from chemists' shops were demonstrated, with all varieties of markings for the various strengths of insulin. It was pointed out that the most desirable syringe for a diabetic was either a one or two millilitre syringe marked with 10 marks per millilitre and no unit markings, so that once the patient had been correctly instructed, both he and his doctor knew exactly how much insulin was being given.

Urine Testing and Renal Thresholds.

All the various modern methods of "tape testing" of urine were shown and their limitations discussed. Stress was laid on the fact that whilst they were very useful means of detecting glycosuria, they were not reliable for quantitative estimations. High and low renal thresholds were discussed. It was pointed out that raised renal thresholds were more common in the older patients, and should be looked for in those patients with peripheral neuritis of obscure ætiology, and in all cases of peripheral vascular disease which were not adequately explained on clinical examination. Lowered renal thresholds were more commonly seen in young patients, and particularly in pregnancy. It was most desirable to establish the threshold by blood and urine sugar estimations in those cases, as attempts to obtain control by urine examination only usually resulted in frequent hypoglycæmic reactions.

Hypoglycæmic Tablets.

The indications for hypoglycæmic tablets were discussed, and their reasonably limited field was emphasized. Case histories were presented which indicated their use and complications. Particular stress was laid on the danger of converting patients from insulin to tablets without very careful control; several patients had become acidotic and one precomatose when insulin was stopped and tablets were started. Stress was also placed on the fact that better control was not achieved by increasing the dose specified for the particular tablets used, while the possibility of severe complications was greatly increased.

Out of the Past.

In this column will be published from time to time extracts, taken from medical journals, newspapers, official and historical records, diaries and so on, dealing with events connected with the early medical history of Australia.

THE RONTGEN RAYS.[1]

[From the *Australasian Medical Gazette*, July 20, 1896.]

A SPECIAL MEETING of the Medical Section of the Royal Society of N.S. Wales was held in the Physics Lecture Room of the University of Sydney (by kind permission of the Senate) on Friday evening, June 19th at 8.15 o'clock. Dr. Robert Scot Skirving the chairman of the section presided. There was a very large attendance of members present, the room being filled. In opening the meeting Dr. Scot Skirving thanked the members for the honor they had conferred upon him by electing him chairman of the section for 1896.

The minutes of the preceding meeting were read by Dr. C. J. Martin, the senior honorary secretary, after which they were confirmed.

The Chairman then briefly introduced the business of the evening, viz., a lecture-demonstration upon "The Rays of Rontgen and their practical application" by Professor Threlfall M.A. and in a few well chosen words introduced the lecturer. Professor Threlfall at once proceeded with the lecture-demonstration.

The practical application of the "Photographic" action of the Radiation in Surgery was exhibited in regard to two patients—(1) a case of osseous disease of the left carpus under the care of Dr. G. T. Hankins; and (2) a case of bullet wound of the right forearm, with lost bullet, under the care of Dr. Frizelle. The left hand, with forearm, of each patient was "photographed", and the plates were inspected at the close of the lecture. The extent of the disease in the wrist was distinctly visible in the radioscript from Case 1, and the situation of the lost missile was plainly denoted in Case 2. Several other cases of radioscripts were exhibited that had been taken by Professor Threlfall.

A very striking practical appreciation of the radiation was shown at the close of the meeting, by giving every one present, who wished it, an opportunity of seeing directly, by the help of a fluorescent screen, the bones of his own hand. A great many availed themselves of the opportunity. Mr. T. A. Pollock, B.Sc., assisted

[1] From the original in the Mitchell Library, Sydney.

Professor Threlfall during the evening. At the conclusion of the meeting, Dr. Scot Skirving moved a hearty vote of thanks to Professor Threlfall for his kindness in placing before the section a very interesting, instructive, and practical lecture-demonstration upon the Röntgen Radiation. Dr. Sydney Jones seconded the motion which was carried unanimously and by acclamation. (Professor Lyle had already on 27 March 1896 given a similar demonstration in Melbourne to members of the Victorian Branch of the B.M.A.)

Special Correspondence.

LONDON LETTER.

BY OUR SPECIAL CORRESPONDENT.

The Nation's Health.

REVIEWING the health of the population in England and Wales for the year 1958 the Chief Medical Officer of the Ministry of Health, Sir John Charles, declares that, until means are found of preventing or improving the treatment of cancer and the degenerative diseases of the circulatory system, the present expectation of life is not likely to be appreciably extended. The expectation of life has remained practically constant for the past five years (69 for males and 74 for females). Diseases of the circulatory system account for over one-third and the cancers for one-sixth of the total deaths. There is little sign of any halt in the rising trend of deaths from lung cancer. On the other hand, maternal mortality reached a new low level (35 per 100,000 births), while infant mortality showed the lowest rate ever recorded in this country. The decline in the incidence and mortality from infectious diseases continued in 1958, with some exceptions. For the first time since 1943, there was a break in the steady decline of diphtheria, a disease which may again become a serious problem unless the level of child immunization is maintained. Scarlet fever and measles were more numerous than was expected. Dysentery also increased and was twice as common in urban as in rural areas.

Diseases of the respiratory system constitute the commonest group of illnesses in general practice in England and Wales, and the common cold has the highest patient casualty rate. It cannot yet be claimed that any virus has been shown to cause the common cold. The recurrent epidemics of respiratory illness which are such a regular feature of the English winter are undoubtedly of mixed origin, but the prospects of unravelling this complex group of illnesses have never been brighter.

The number of cases of early syphilis attending clinics continued to decline although not consistently throughout the country. A sharp rise in the number of new cases of gonorrhœa in both sexes gives cause for anxiety, being most marked in London and other areas where immigrants from overseas are arriving. Treatment with penicillin often fails, even when given in large doses, and a Committee of the Medical Research Council is investigating the problem of sensitivity of the gonococcus.

The Risks of Progress.

A subject which is rarely faced and discussed is the ultimate effect of the advancement of medicine and science on the human race. It can be seen superficially as the conquest or control of certain diseases, but also as the advance and spread of others—e.g., leukæmia, cancer and coronary disease. The deeper aspects of this subject were outlined recently by Lord Adrian, O.M., F.R.S., in the Fawley Foundation Lecture at Southampton University. He commented: "We have aimed and must aim, at keeping alive every child that is born but we have succeeded so well in diminishing early mortality that we are certainly preserving many unfavourable genes which would otherwise have died out. If we set out to save the unfit, we must expect more unfitness in the world and more inheritance of the factors which promote it." He suggested the necessity for repeated health surveys, without which we should never be rid of the fear that our progress was making the world a more dangerous place to live in. Even so, genetic deterioration might make the human race less able to live in the world, even though its dangers were not allowed to increase. He was not too pessimistic as to the future. Medicine advances quite as rapidly as any other branch of applied science.

Professor P. B. Medawar, Professor of Zoology and Comparative Anatomy in the University of London, speaking in the second Reith Lecture on the "Future of Man" on the B.B.C.'s Home Service Programme, dismissed the idea that advances in medicine and hygiene were undermining the general fitness of the human race. He declared that such an idea was based on the belief that there was an hereditary or genetic element in all human ills and disabilities, although it might amount to no more than a predisposition. It could be argued that because of the discoveries of insulin and antibiotics, etc., we were preserving for life and reproduction people who even 10 years ago would have died. We were, therefore, preserving the genetically ill-favoured, the hereditary weaklings, who could intermarry and therefore undermine the constitution of normal people, and, as a result, mankind was going downhill. If by "going downhill" was meant decline in biological fitness with the implication that mankind would probably die out, then the arrangement was simply a museum of self-contradictions. What was more to be feared was a slow decline in human intelligence. If that was happening at all, it was because the rather stupid were biologically fitter than those who were mentally more intelligent, not because medicine was striving to raise the biological fitness of those who otherwise might be hopelessly unfit.

Correspondence.

THE FUTURE OF THE AUSTRALIAN ABORIGINAL.

SIR: With reference to Professor Cleland's article on "The Future of the Australian Aboriginal", I read this with great interest and applaud its sentiments; but I cannot agree in giving most importance to nature as opposed to nurture. The dominating influence of nurture (or culture) can be felt in our own case. I think Professor Cleland will agree that an English culture ruled in white Australia for over a century after settlement, and is still very strong. Can we expect a quicker change from the aborigines?

They are culturally "food gatherers", knowing nothing of cultivation. Other peoples in this stage came in contact with white civilization in other countries long before the nineteenth century; but I have not been able to trace any one of them which has been received into civilization in the way we desire. The rule (for which I take no responsibility) seems to be, so far, that if a primitive people has previously practised cultivation before coming into contact with white civilization, it has some hope of survival; but if not, not.

If they are to survive as civilized aborigines, they must be incorporated in sufficient numbers to prevent loneliness, and there is no sign of this. If they are to be "bred in", it can be done by rearing their children in an exclusively white environment (which will not please the parents), or by cross breeding. As white women do not greatly want to go out to the pastoral areas and marry stockmen of any colour, this means white fathers and black mothers—a scheme not likely to produce permanent satisfaction to many people, and likely to produce too large a class of discontented and troublesome young blackfellows.

I deeply regret that for these reasons I can see little hope of large-scale integration on the lines laid down. I sincerely hope that I will be shown my error.

Yours, etc.,

J. B. HOGG.

44 The Avenue,
Nedlands, W.A.
December 31, 1959.

SIR: Professor Cleland's statement on "The Future of the Australian Aboriginal" under "Points of View" (January 2, 1960) calls for comment. In discussing the *Aborigines Act* of South Australia, 1934-1939, he fails to mention that everyone in South Australia with any aboriginal blood is legally under the control of the *Aborigines Act*, and can only acquire complete legal status of the white community by applying for exemption. The applicant is then informed that if, after a period of three years, character and conduct prove exemplary, exemption will be granted. And now it is even proposed to make exemptions revokable. The certificate with photograph is supposed to be carried on the person for life. Many regard the whole process as an insult and refuse to apply.

I submit that people who have been educated compulsorily in our State schools, are working in our midst and paying income tax should be citizens in their own right.

Professor Cleland's comments on the repeal of the Consorting Clause of the *Police Act* are difficult to understand. The Consorting Clause was repealed by both Houses of Parliament. The public, stirred by police questionings and interference with legitimate friendships between white and native, was overwhelmingly in favour of the repeal. "Unwise propaganda, misleading statements and misapplied sympathy" were not in evidence. Professor Cleland states that the clause was inserted "to enable the situation to be controlled, especially in the far interior, where undesirable doggers and swagmen may take native women as temporary mistresses"; but Section 34 (a) of the *Aborigines Act*, 1934-1939, provides for this and operates if required. Today there are no doggers in the tribal north-west of South Australia. They decamped subsequent to the founding of the Ernabella Mission in the Musgraves.

Professor Cleland prefers the term "part-white" to "part-aboriginal"; but these people are Australians and need no specific title, and few need special treatment.

It is correct that a well-made wurlie gives better protection than a tin shed. Why, then, in 1959 were three tin sheds built at Oodnadatta by the Aborigines Protection Board, of which Professor Cleland is Deputy Chairman? The sheds are 9 feet by 7 feet, open at one side, and with earth floor. In September I found them too cold to stand in.

In speaking of citizenship, Professor Cleland states that even a minor enjoys the same protections as enrolled citizens. He is right; and when in the near future dark Australians are citizens, those in tribal territory, although not enrolled, will enjoy the same protection as enrolled citizens. I agree heartily that "it would not be in the interests of the natives for them to become the idle protégés of a Welfare State"; but is sufficient care being taken to prevent this at the present time? Dark Australians are morally a Commonwealth responsibility, and the Federal Government which sponsored assimiliation is paving the way to citizenship.

It is very regrettable that Professor Cleland does not grant good faith to many who do not see eye to eye with him on the *Aborigines Act*. To refer to efforts of conscientious people seeking an improvement of the *Aborigines Act* of South Australia, 1934-1939—the oldest in the Commonwealth—as providing "capital for attacking the British Commonwealth" is as unworthy as it is untrue. Surely this was not a considered statement.

Yours, etc.,

CHARLES DUGUID.

Adelaide,
South Australia.
January 3, 1960.

SIR: I read with considerable interest Professor Cleland's thoughtful note on the future of the Australian aborigines. In the main I am in agreement with what he says, but I should like to make a few comments.

Professor Cleland implies that the brain of the aboriginal works differently from that of the European—the aboriginal is mentally adapted to pastoral pursuits, the European has inherited an "engineering" type of brain. Quite apart from the suggestion here of inheritance of acquired characteristics, of which no valid evidence has ever been presented, there is nothing to indicate that the aboriginal, with proper training, would not make as good an engineer as anybody else. Indeed, I have known several who were excellent motor mechanics, carpenters, etc. Professor Cleland does admit that the aborigines may have a latent mechanical ability which may fit them for looking after farm machinery and station garages. But why stop there? If aborigines can get that far—and a great many now are at that stage—they can certainly go farther in the same direction when they are given the encouragement and training to do so.

An objection raised against employment of aborigines is their lack of "stickability" in any job. This is open to considerable doubt. In any case, if it refers to the tendency of semi-nomadic aborigines to go "walkabout" for a few weeks every now and then, I would ask in what way this differs from the general and accepted propensity of Europeans also to go "walkabout" for a few weeks every year—on their annual holidays. The habits of the two peoples in this respect are strictly comparable; in each case there is a yearning to leave the humdrum everyday working life and go somewhere else "closer to Nature"—often at considerable personal inconvenience, whether by foot, car or caravan.

There is one further point: when we talk of assimilating the aborigines, we usually visualize a process of physical absorption, with the aboriginal characters becoming progressively more and more diluted in successive generations until they disappear entirely into the European population. There is not the slightest doubt that that can happen very readily, and large numbers of people with aboriginal ancestry have passed over completely and indistinguishably into the non-coloured side of the population. However—and this must be a shock to some Europeans—that is not what the average aboriginal wants. He does not accept the implication of inferiority. He is as proud of his colour and his people as is the European, and does not regard the title "blackfellow" as opprobrious. What he wants is to retain all the ethnic characteristics of which he is so proud with the right to enjoy all the privileges of the "whitefellow". His complaint is solely that this last is denied him. The fact that full enjoyment of those privileges must continue to be denied to large numbers of aborigines—many still barely out of a nomadic existence—for some time yet is an unfortunate fact that doctrinaire sociologists with no practical knowledge of the real problem must learn to accept.

I endorse completely Professor Cleland's commendation of the efforts of the Commonwealth and State Governments to cope with this very difficult situation. As one result of their efforts the aborigines are on the increase. I once heard a Maori claim that ultimately the Maoris would absorb the Europeans in New Zealand. It is unlikely that the aborigines will expand to that extent in Australia, but the sentiment is not out of keeping with the aborigines' spirit. Certainly it is a point of view that might give the European some cause for thought.

Yours, etc.,

A. A. ABBIE.

Department of Anatomy and Histology,
University of Adelaide,
Adelaide.
January 7, 1960.

SIR: I commend the article on this subject (MED. J. AUST., January 2, 1960) in most of its practical suggestions for immediate action. I deplore some of the theoretical points on which long-range decisions must be based.

I contend that nurture is the chief factor in our mental equipment, not nature. There is little possibility of social acceptibility anywhere for a European baby nurtured by a wolf, even though his natural capacity reached genius level. The rare aboriginal child genuinely nurtured as their own by European parents has exactly the propensities of the adoptive parents, and one of them is a bachelor of arts, who would doubtless be quietly amused at the suggestion that he was more prone to the "call of the wild" than I am.

Most of us have seen animals "who" are "almost" human in temperament because some human foster-parent has nurtured them from a helpless age.

There is less difference in instinct or inborn capacity between differently-coloured members of the human species than there is betwen Dalmatians and blue cattle-dogs, which are separated by hundreds of generations of deliberate selective breeding and specialization. Many anthropologists place Caucasian (Indo-European or "Ayran") stock, which predominates in Australia, closer to the original Australians than to Africans, Pacific Islanders or Mongolian stocks.

The restriction of aboriginals to the most isolated and menial of all jobs as listed in your article may be a fact, but is still due to white-skinned prejudice, not to inherent aboriginal backwardness. A local school-teacher told me one of the aboriginals in his class is the brightest pupil, and in general there is little difference in academic capacity between children of different pigmentation. I asked what would happen to the lad who topped his class. "He'll probably get a job on a station or fettling." Why so? "There are no opportunities for him in the business world. Most aboriginals reaching puberty lose interest in studies and get restless. Like all adolescents, they want to be acceptable as adults. They are accepted as equals by white children, not adults." Scant wonder, then, that the aboriginal nurse-maid, so refined and demure, shows her "ingratitude" by going walkabout with some shiftless male in a bark humpy with dirt, flies and poverty when

she reaches adolescence. She has learnt who will accept her as a woman, not a rouse-about.

The extra legal privileges of aborigines listed in the article seem to be almost inevitable for continued tribal survival (exemptions from hunting restrictions, etc.) and surely would in practice be extended to any white man living as a tribal aboriginal. The exemptions are not so much favours as a residue of restraint in wresting the sorry remnants of their heritage from the natives who preserved it for us.

The fact that an occasional capital crime occurs due to alcohol inside substantially Australoid persons is no argument for alcoholic apartheid. Are aborigines more prone to alcoholism, to crime or to grave offences while drunk than people with Irish accents, unshaven chins, dirty athletic singlets or bare feet? Your author would brand a European who cannot take his liquor. It is possible—just tattoo a black A on his nose. But the accent and other stigmata of the downtrodden I have listed would, I estimate, be about as reliable statistically as an index of proneness to alcoholic violence as a sombre skin, and as easy to identify with assurance. Many hotels in America exclude Jews as effectively as Negroes or people without tie and coat.

A local aboriginal told me of a man of Indian extraction who spoke more languages fluently than I do (and so do many aborigines), gently but firmly refused a drink in this city in spite of explanations and representations by his friends and eventually trades union officials. The then acting police chief finally negotiated a compromise with a particular publican in such a way that the well-pigmented gentleman could consume his liquor without being perceived by other darker-tinted people.

If we must license dark skins to contain alcohol, let us at least renounce the master-race doctrine of white supremacy which fouls our national repute in the councils of the world, and make everyone produce his licence to drink. Could there be a surer way to reduce the ever-growing menace of our number one public health problem? All the multitude of other liquor restrictions might then become unnecessary, and it would be no more hardship than the compulsory carrying of a car driver's licence, which I understand is the new law in this State—especially if all types of licences were incorporated on one small convenient form, for economy of recording as well as convenience to the licensee.

Yes, there is an aboriginal really successful in the higher walks of life. I have seen and heard him in the flesh—Harold Blair, singer.

The social problem of less-whites is a problem mainly because their parents were outcasts. There is a tendency for people who are already outcasts amongst "full-bloods" (white or dark) to contract a disapproved type of marriage (in a non-accepted, non-accepting social group). All sorts of social misfits are known to seek out other sorts of misfits in marriages. We have our bohemian communities and ghettoes. It is not the mixing of bloods or genes that does the damage, if we can judge by the great charm and ability of the offspring of miscegenous marriages in accepting communities (New Zealand, Hawaii, Shanghai). If we cannot judge the beneficial effect of full racial tolerance in such communities, where can we judge it?

It is not enough to have several governments exploring different costly policies all based on the White Australia myth. If we make any progress in such experiments in any State or Territory, it is to the extent that we treat part or whole aborigines as humans with the same needs and faults, rights and duties as whole or part palefaces, with the probable exception that the latter group will probably require more dermatological care in our climate.

The cohabiting of diggers, saints or swagmen with temporary mistresses anywhere with or without exploitation or supplying of liquor is doubtless often sinister or otherwise undesirable, and there are clean, tidy, racial-hygiene-minded people who would forbid it between people of all hues if they could "catch such a rascal *in flagrante delictu*"; but to "do" it (surely just as sinister) by means of Police Acts, not mentioning the far interior, the swagmen, the liquor, the exploitation and all the rest, is not only bungling law, like our immigrants' dictation test, but is just the sort of jackboot and sjambok policy that characterizes "*Mein Kampf*" and apartheid, no doubt with the highest motives, and the greatest confusion of aims.

Yours, etc.,

DOUGLAS EVERINGHAM.

P.O. Box 328,
Rockhampton, Queensland.
January 8, 1960.

AGING OF THE AUSTRALIAN POPULATION.

SIR: From time to time attention is drawn to the age distribution of the Australian population. In company with many countries throughout the world, Australia has experienced the process, known widely as aging, whereby there is now a greater proportion of old persons in the population than formerly. Table I gives an idea of this change in Australia from 1871 up to the most recent time, 1958.

TABLE I.

Population: Proportional Age Distribution, Australia.

(From "Year Book of the Commonwealth of Australia", Number 43.)

Census.	Sex.	Proportion of Persons at Ages (Years)		
		0 to 14.	15 to 64.	65 and Over.
1871	M.	38·8	59·1	2·1
1881	M.	36·4	60·8	2·3
1891	M.	34·8	62·0	3·2
1901	M.	33·9	61·8	4·3
1911	M.	30·9	64·8	4·3
1921	M.	31·6	63·9	4·5
1933	M.	27·5	66·1	6·4
1947	M.	25·5	67·1	7·4
1954	M.	28·8	63·8	7·4
(1958)[1] ..	M.	30·2	62·5	7·3
1871	F.	46·0	52·6	1·4
1881	F.	41·9	56·0	2·1
1891	F.	39·4	58·1	2·5
1901	F.	36·5	59·9	3·6
1911	F.	32·5	63·3	4·2
1921	F.	31·8	63·8	4·4
1933	F.	27·4	66·0	6·6
1947	F.	24·6	66·7	8·7
1954	F.	28·2	62·5	9·3
(1958)[1] ..	F.	29·5	60·9	9·6

[1] Estimated.

This change is almost universally ascribed to changes in mortality, although the Royal Commission on Population (1949)[1] was at some pains to counteract this impression; "Population Studies" (1953)[2] and Lancaster (1954)[3] set up life table models showing that mortality had relatively little effect. The most recent changes in the Australian population would also point in this direction, as with continually improving mortality the changes in the proportion of the aged have been slight, and there has been even a tendency for the proportion to fall for the males.

The age group 15 to 64 years may be regarded as containing most of the bread-winners. The proportion in this age group has remained relatively stable, varying between 59·1% in 1871 and 67·1% in 1947. The high figure in 1947 was due principally to the low birth rates of the thirties giving an unduly small number of children for 1947.

The elderly present special problems, medical and economic; the medical problems of hospitalization are very important, and the provision of pensions requires either saving or taxation. We can see whether mortality changes put undue economic burdens on the population by considering what proportion of their lives will be spent in old age by actual generations (or cohorts) as in Table II, where, using the generation life tables of Lancaster (1959),[4] average proportions of each person's life spent in three age groups are given. Males born in 1851 would have spent some 10·5% at ages over 65 years, whereas (using some minor extrapolations) those born in 1891 would have spent 11·7%. The corresponding proportions for females are 13·3% and 15·6%. Mortality changes have, of course, affected these proportions; but the changes in the proportions in the generations are small compared with those in the actual population given in Table I. The point here is that mortality changes have not affected the ability of a generation to save for its old age, whether in the form of insurance premiums or savings in a more indirect form such as in industrial investment.

[1] Report of the Royal Commission on Population (1949), H.M. Stationery Office, London.

[2] "The Aging of Populations and Its Economic and Social Implications" (1953), Population Studies, Number 26, United Nations, Geneva.

[3] MED. J. AUST. (1954), 2:548 (October 2).

[4] *Aust. J. Statist.* (1959), 1:19.

Migration has a twofold effect on age distribution. First, migrants are usually young adults and their children, and second, after arrival, their birth rates, being young adults, are higher than those of the general population.

There is some reason to believe that invalidity at ages 15 to 64 years has decreased with the gradual disappearance of tuberculosis; but some doubts remain about the position at ages over 65 years. There is a widespread view which suggests that a larger proportion of the elderly should be infirm because they have been kept alive at lower ages. Against this we can note that only in the infectious diseases has real progress been made, and we may look forward to periods where less of the elderly will be survivors from any overt clinical form of tuberculosis, for example.

TABLE II.

The Proportion of Life Spent in Each Age-Group.

Generation Life Tables for those Born in the Year.	Sex.	Percentages at Ages		
		0 to 14.	15 to 64.	65 and Over.
1851	M.	24·7	64·8	10·5
1861	M.	25·0	64·4	10·6
1871	M.	24·1	64·7	11·2
1881	M.	23·4	65·1	11·5
1891	M.	22·9	65·4	11·7
1851	F.	23·5	63·2	13·3
1861	F.	23·5	63·0	13·5
1871	F.	22·6	63·1	14·2
1881	F.	22·1	63·0	14·9
1891	F.	21·6	62·9	15·6
Calendar life tables:				
1921 ..	M.	22·9	66·0	11·1
1921 ..	F.	21·8	64·5	13·7
1954 ..	M.	21·6	66·2	12·2
1954 ..	F.	20·1	63·7	16·2

The demographic history of Australia is such that many factors are responsible for the form of the age distribution at any time or over a period. For example, only 1·4% of females were over the age of 65 years in 1871. This was due to a low rate of migration of females in the earliest years of the Australian Colonies. In more recent times, most Australian citizens are Australian born, and so the elderly in 1958 reflect the approximate equality of the sexes at birth and the more favourable mortality rates for females at all ages, including, of course, the years of the first World War.

In conclusion, we may express the opinion that the major changes in age distribution in Australia have been due to changes in fertility and, to a lesser degree, in immigration. Australia in the past has had less than its share of old people. If fertility ever settles down to be just sufficient for maintenance of the population, we may expect about 12% of males and 16% of females to be over the age of 65 years. In any case, we can expect the proportion of persons at ages 15 to 64 years to remain at approximately 60% to 66%.

Yours, etc.,

H. O. LANCASTER,

Department of Mathematical Statistics,
University of Sydney,
Sydney.
December 16, 1959.

TETANUS PROPHYLAXIS.

SIR: Dr. K. D. Murray's letter of September 23, 1959, has stimulated me to write on tetanus prophylaxis. There appears to be a general timid swing away from the use of A.T.S. (tetanus antiserum), substituting for it tetanus toxoid.

I recommend to all interested that invariably in place of A.T.S. 1500 units (or under), tetanus toxoid be given (1 ml.). The reasons prompting this suggestion are: (i) 1500 units of A.T.S. is completely inadequate; (ii) it can be dangerous; (iii) tetanus toxoid is safe. It can be given without sensitivity test. It is my opinion that it gives as much protection, or more, than 1500 units of A.T.S.

I base my conclusions on the fact that I have used this method on over 3000 cases of penetrating wounds. No tetanus developed in any of these.

Commonwealth Serum Laboratories state: "A single injection of tetanus toxoid in a previously unimmunized person confers no immunity whatsoever." This is an illogical statement. It contradicts the laws of immunity.

Since 1943 I have made it a practice of giving tetanus toxoid (1 ml.) to all cases of penetrating or dirty wounds. I cannot say what percentage of these had previously been actively immunized. It would be less than 20%. The only exceptions I make are those who had completed a course of immunity within 18 months.

Severer wounds with shock and tissue destruction naturally require treatment with A.T.S. (tetanus antiserum) in large doses.

Yours, etc.,

TONY WALSH.

Northam,
Western Australia.
January 1, 1960.

HYPOTHERMIA IN CARDIAC SURGERY.

SIR: I was interested to read Mr. Harry Windsor's views on certain aspects of cardiac surgery in your correspondence column of December 26, 1959.

In the pædiatric age group, there would appear to be a very definite place for pulmonary valvotomy through the pulmonary artery. To open the right ventricle in these cases is not only unnecessary, but could be harmful, since this chamber is already under considerable strain and does not take kindly to the further insult of ventriculotomy. It is agreed that the problem may well be different in adult cases with long-established thickening of the infundibular muscle. This is a strong argument for dealing with these cases at an earlier age.

It may well be that all atrial septal defects will ultimately undergo repair on a pump oxygenator once the safety of the pump is comparable with that of hypothermia. The risks as set out by Mr. Windsor are not very convincing; the diagnosis should not be inaccurate, the presence of anomalous veins presents no particular problem, and where any doubt exists as to the possible presence of a "primum" defect, the pump should always be employed. If hypothermia is combined with coronary perfusion, ventricular fibrillation will rarely be a problem, and the suturing of the defect need not be hasty or inaccurate, since up to fifteen minutes is available if required.

It would seem most unwise to leave an atrial septal defect of any size unrepaired until the age of twenty, since it can be repaired with much greater safety during childhood. Surely any cardiac surgeon of experience would agree that the technical difficulties are very much greater, whether hypothermia or by-pass is utilized, over the age of 10 to 12 years.

Yours, etc.,

DOUGLAS COHEN.

"Wyoming",
175 Macquarie Street,
Sydney.
January 5, 1960.

SIR: In reply to Dr. Harry Windsor's letter (MED. J. AUST., December 26, 1959), I should like to point out that a full discussion on the selection of such cases, of which he was so critical, was made by Dr. Douglas Cohen on pages 788 and 789 immediately following my presentation.

"Poor risk" tetralogy of Fallot is not uncommon in early childhood, but is a condition rarely encountered by those engaged in adult surgery, simply because they die when very young. Hypothermia is used to make a Blalock procedure safer in such cases, and this enables them to live and grow and reach an age when a curative operation under total by-pass may be considered. A curative procedure in early childhood has resulted in a prohibitively high mortality in those few centres overseas where it has been attempted. No one is more interested or desirous of overcoming the technical difficulties and problems associated with cardiac by-pass in this early age group than we are at the Royal Alexandra Hospital for Children, and, indeed, we are working strenuously to this end.

Dr. Windsor wondered if it is right to subject children to operation under hypothermia suffering from atrial septal

defect—septum secondum—as "most of these children are asymptomatic and one can almost guarantee that at the age of twenty they will still be that way". He must be aware of the fact that 10% of such children will develop pulmonary hypertension during that interval. Why let the heart thrash itself to death with a big shunt, and why wait till serious effects are produced before attempting closure of the defect? Why wait till the mortality and morbidity are so much higher and the economic and social disruption so much greater? Why wait, when it can be undertaken so successfully in childhood with a mortality of 2%? Surely it is wiser to operate on these patients when children and let them grow normally.

At the moment, cardiac by-pass procedures involve considerable morbidity and a significant mortality even in the world's best units. Any cardiac unit which has developed a well-controlled technique for hypothermia, involving virtually no risk whatever, must have very strong and clear-cut indications in favour of abandoning it for the other.

Most, if not all, congenital heart surgery should be undertaken in childhood, and hypothermia, I believe, Sir, is still making history.

Yours, etc.,

VICTOR HERCUS.

Congenital Heart Disease Unit,
Royal Alexandra Hospital for Children,
Sydney.
January 6, 1960.

MEDICAL RESEARCH IN AUSTRALIA.

SIR: It is evident that the medical profession will be asked to support the appeal for research funds by the Heart Foundation of Australia during 1960. Heart disease is an important cause of death and disability, and research at every level will help to combat it. Money is one of the greatest needs for medical research, but it is not the only one, and I should like to suggest the special need at the present time—i.e., before the appeal is launched—for taking stock of the organization of medical research in Australia.

Past experience makes it more than possible that a multiplication of independent bodies making grants for research will result mainly in increasing the number of people doing second-rate research on uncoordinated projects. Our human resources in Australia are limited, and the quality and significance of the work on which men with a flair for investigation are employed is much more important than the over-all sum of money provided for research. The whole pattern of medical research and of the social requirements of medicine is changing too rapidly for committees of the type that must control such funds to give any guidance on the way lines of investigation should develop.

It is urgent, in my opinion, that some small, authoritative group should consider the structure and support of medical research in Australia, and make its recommendations before new complexities are added. The Murray Commission has been of great value to the universities, and something broadly equivalent to this is badly needed in the field of medical research. Such a commission would be in a position to hear all views and seek the most effective solution for Australian conditions. My own views may be too ambitious; but as an indication of how one experienced worker is thinking, I shall state them briefly.

There are three separate aspects of medical research:

1. Research directly associated with university teaching—i.e., the investigational activities of university staff members that is needed to maintain vigour of teaching, and the training of Ph.D. students. This should be the unqualified responsibility of the universities themselves, and supported by funds from the University Grants Commission. All other funds going to university departments for research should be distributed through the university's general funds for medical research.

2. Basic research in biological science, which will always be centred on the work of gifted individuals. This should be the responsibility of an organization equivalent to the Medical Research Council of Great Britain, with an executive head of high status and a supporting committee chosen only for their qualifications to assist in maintaining the status and integrity of science. There are precedents for making this part of C.S.I.R.O. and for including within its responsibility the John Curtin Institute of the Australian National University. It should support basic research in the universities in the form of full-time units, each under the scientific control of an investigator of established effectiveness. These would be analogous to the units adopted by Sir Edward Mellanby as the standard method of supporting research in Great Britain, and they should be free of any dependence on general university research funds.

3. Post-graduate specialized training and clinical research, whose role in both instances is the preparation of young medical graduates for an effective professional career. This is the sphere in which the medical profession and especially the Royal Colleges should find their major responsibility. Here the indefinite boundary line between training and research must be accepted. I should lean strongly toward regarding all such appointments as for training until an individual showed unmistakably his effectiveness as an investigator. He should then be given appropriate opportunity and support exactly equivalent to that suggested for basic research under group 2. To make the best use of present funds in the clinical field, I believe that there should be a means of pooling the resources of existing bodies supporting clinical research so as to make the maximal contribution to top-level clinical training. A special committee appointed by the Royal Colleges and working in association with the executive head under 2 might be appropriate.

Yours, etc.,

F. M. BURNET,
Director.

The Walter and Eliza Hall Institute of Medical Research,
Melbourne.
January 5, 1960.

CHILDREN'S MEDICAL RESEARCH FOUNDATION OF NEW SOUTH WALES.

SIR: During the past sixteen months, the medical profession of New South Wales has very generously subscribed more than £10,000 to the Children's Medical Research Foundation of this State. Nearly 90% of these donors have subscribed on two or more occasions, and a significant number of them have also promised further donations.

May I take this opportunity of expressing my most sincere gratitude for this magnificent response on the part of the medical profession of this State?

Yours, etc.,

LORIMER DODS,
Chairman.

Executive Committee,
Children's Medical Research Foundation Appeal,
Royal Alexandra Hospital for Children,
Camperdown, N.S.W.
December 30, 1959.

UNEXPLAINED SUDDEN DEATH IN YOUNG ADULTS.

SIR: Your "Current Comment" paragraph of January 2, "Unexplained Sudden Death in Young Adults", brought to mind such a case five years ago, in which I had the possibly unusual experience of witnessing immediately after death occurred.

The patient was a young married woman of 21, well nourished and mother of two children, one a baby of six months. She was doing her usual household jobs one Saturday afternoon when she, as her relatives thought, "fainted" at the clothesline. By one of those coincidences I was on a routine visit to the house to see the patient's nieces, who had mumps, and called just as they were carrying her on to a bed. Her colour was normal, pupils large, limbs, etc., flaccid, no alimentary canal or bladder actions. I gave adrenaline, "Coramine" and artificial respiration without avail.

That she had appeared to enjoy her usual health for the week or two before death I know, as I had visited the house several times in that period for other members of the family, sometimes seeing the patient in passing. Also, on the Saturday afternoon in question, she had even had a bet on the races. I understand the cause of death was reported by the coroner as "myocarditis", after autopsy including gastric analysis.

Her husband volunteered to me the information that she probably had, recently before death, been taking "pills" from the chemists, possibly in excess of "recommended" doses, as she had a dread of further pregnancy (her youngest child was six months old).

Yours, etc.,

21 High Street,
Prahran,
Victoria.
January 9, 1960.

H. H. WILSON.

Post-Graduate Work.

THE POST-GRADUATE COMMITTEE IN MEDICINE IN THE UNIVERSITY OF SYDNEY.

Tape Recordings.

THE Post-Graduate Committee in Medicine in the University of Sydney announces that the following tape recordings, made from broadcasts over the Voice of America Forum-Medicine Programme, have been lent to the Committee by the United States Information Service for a period of several months. These tape recordings are now available for loan to approved medical groups on application to the Post-Graduate Committee in Medicine, 131 Macquarie Street, Sydney.

"Is Biophysics the Ultimate Science?": playing time, 30 minutes; Dr. Howard L. Andrews, Chief of the Nuclear Radiation Biology Section of the National Institutes of Health.

"Health and Safety in the Atomic Energy Programme": playing time, 30 minutes; Dr. Shields Warren, Professor of Pathology, Harvard University, and Pathologist, New England's Deaconess Hospital, Boston.

"Trends in Medical Education in the United States": playing time, 30 minutes; Dr. Edmunds G. Dimond, Professor and Chairman of the Department of Medicine, University of Kansas School of Medicine.

"Pharmaceuticals", with script: playing time, 30 minutes; Dr. John T. Connor, President of D. E. Merrick & Co., Inc., of Ralway, New Jersey.

"Communications": playing time, 30 minutes; Dr. Schafer.

"Rehabilitation", with script: playing time, 30 minutes; Professor Howard A. Rusk, Chairman of the Department of Physical Medicine and Rehabilitation, New York University Bellevue Medical Centre.

"Interpersonal Relations in Medicine", with script: playing time, 30 minutes; Dr. Albert J. Glass, Chief of the Division of Neuro-Psychiatry, Office of the Surgeon-General of the U.S. Army.

"The Medical Story of Man in Space": playing time, 30 minutes; Dr. William Randolph Lovelace, Chairman of the Special Advisory Committee on Life Sciences of the National Aeronautics and Space Administration.

"Cancer and the Mystery of Growth": playing time, 30 minutes; Dr. George Crile, jr., Chief of the Department of Surgery, Cleveland Clinic Foundation, and the Frank E. Bunce Educational Institute, Cleveland, Ohio.

"Stress and Adaptation": playing time, 30 minutes; Dr. Hans Selye, Director of the Institute of Experimental Medicine and Surgery, University of Montreal.

"Cardiovascular Disease": playing time, 30 minutes; Dr. Paul Dudley White, one of the United States' foremost cardiologists.

"Recent Developments in the Field of Surgery": playing time, 30 minutes; Dr. M. E. DeBakey, Chairman of the Department of Surgery at the Baylor University College of Medicine, Houston, Texas.

"The Evolution of Primary Aldosteronism": playing time, 30 minutes; Dr. Jerome Conn, one of America's foremost specialists in the field of medical biochemistry.

"Psychiatry at Mid-Century": playing time, 30 minutes; Dr. David Rioch.

"Epidemics in Our Time", with script: playing time, 30 minutes; Dr. Jonas Salk, Director of the Virus Research Laboratory, University of Pittsburgh School of Medicine.

"Horizons in Medicine": playing time, 30 minutes; Dr. Paul W. Schafer, Executive Director of the Television Division, Walter Reed Army Medical Center, Washington.

Notes and News.

Northern Territory Chest Survey.

The Federal Minister for Health, Dr. the Hon. D. A. Cameron, stated recently that during the recently completed chest X-ray survey of the Northern Territory, the Northern Territory Medical Service had received invaluable assistance from the Anti-Tuberculosis Association of New South Wales. A total of 19,028 residents of the Territory had been examined radiologically; about half of these were of aboriginal or mixed blood, the remainder being of European origin. X-ray teams, including a medical officer of the Anti-Tuberculosis Association, had visited every locality in the Territory to which it had been possible to transport X-ray apparatus. The R.A.A.F. and other units of the Armed Services had assisted in overcoming the formidable transport problems.

Dr. Cameron said the survey had again demonstrated the suitability of modern 70 mm. mirror-camera photofluorographic X-ray units, even when operating in hot and distant localities. As well as revealing the presence of unsuspected pulmonary tuberculosis, the chest films had also assisted in the diagnosis of many other diseases and abnormalities of the chest. The incidence of tuberculosis found was less than expected. Every resident of the Northern Territory, who needed it, would be offered treatment or further investigation by the Northern Territory Medical Service. Tuberculosis allowances would be available to those who were eligible, and were found to be suffering from infectious tuberculosis. The survey had presented a challenge. Its successful completion marked a notable advance in the national campaign to eradicate tuberculosis from all sections of the Australian community.

The World Medical Association: Fourteenth General Assembly.

The German Medical Association, host of the Fourteenth General Assembly of the World Medical Association scheduled to convene in West Berlin from September 15 to 22, 1960, has extended a cordial invitation to all the doctors of the world to attend this outstanding meeting.

The *Bundesärztekammer* (German Medical Association) will convene its 1960 Annual Meeting concurrently with the convening of the General Assembly. This is the first time that a host medical association to the W.M.A. has scheduled its own annual meeting in conjunction with the meeting of the W.M.A. The two associations will meet jointly in their opening and closing plenary sessions. All activities, including the post-graduate teaching film programme, exhibits and scientific sessions, will be held in one building, in order to enable doctors to attend the various sessions associated with each meeting which are of special interest to them.

Additional information, including programmes and schedules, will be available on or about March 1, 1960, at W.M.A. Headquarters Secretariat. Applications should be addressed to The World Medical Association, 10 Columbus Circle, New York 19, New York.

Naval, Military and Air Force.

APPOINTMENTS.

THE following appointments, changes, etc. are published in the *Commonwealth of Australia Gazette*, No. 76, of December 3, 1959.

AUSTRALIAN MILITARY FORCES.

Australian Regular Army.

Royal Australian Army Medical Corps (Medical).

To be Temporary Major, 23rd October, 1959.—2/12032 Captain C. N. Matthews.

Citizen Military Forces.

Northern Command.

Royal Australian Army Medical Corps (Medical).—1/39219 Captain (provisionally) J. S. Harte relinquishes the provisional rank of Captain, 15th October, 1959, is transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical)) (Northern Command) and is granted the honorary rank of Captain, 16th October, 1959.

To be Major, 5th November, 1959—1/67935 Captain (Temporary Major) J. A. Nye.

Eastern Command.

Royal Australian Army Medical Corps (Medical).—2/130107 Captain C. B. Saunders ceases to be seconded whilst in the United Kingdom, 31st August, 1959. 2/130107 Captain C. B. Saunders is transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical)) (Eastern Command), 1st September, 1959. To be Major, 6th October, 1959—2/79254 Captain R. J. M. Atkinson.

The provisional rank of 2/79296 Captain J. C. Crakanthorp is confirmed. 2/146624 Captain (provisionally) R. G. Congdon relinquishes the provisional rank of Captain, 29th September, 1959, is transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical)) (Eastern Command), and is granted the honorary rank of Captain, 30th September, 1959. 2/147990 Captain (provisionally) P. P. Manzie relinquishes the provisional rank of Captain, 22nd October, 1959, and is transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical)) (Eastern Command) in the honorary rank of Captain, 23rd October, 1959.

Southern Command.

Royal Australian Army Medical Corps (Medical).—The provisional appointments of the following officers are terminated:—Captains 3/87648 I. C. Goy, 22nd March, 1959, and F3/1036 D. B. Newton, 3rd September, 1959. F3/1030 Captain A. K. Garven is transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical)) (Southern Command), 25th September, 1959. To be Captains (provisionally)—3/87648 Ian Christopher Goy, 23rd March, 1959, and F3/1036 Dorothy Barbara Newton, 4th September, 1959.

The following officers are transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical)) (Southern Command), 14th October, 1959:—Captains 3/50266 F. P. Callaghan and 3/139331 H. W. Hardy.

Central Command.

Royal Australian Army Medical Corps (Medical).—4/32035 Captain R. Britten-Jones is seconded whilst in the United Kingdom, 1st May, 1956.

4/31905 Major G. W. E. Aitken is appointed to command 1st Casualty Clearing Station, and to be Temporary Lieutenant-Colonel, 1st October, 1959.

Western Command.

Royal Australian Army Medical Corps (Medical).—The provisional rank of 5/26587 Captain R. L. Bissett is confirmed. To be Major, 20th October, 1959—5/26514 Captain (Temporary Major) R. Paton.

To be Major, 28th October, 1959—5/26531 Captain (Temporary Major) M. N. I. Walters.

Reserve Citizen Military Forces.

Royal Australian Army Medical Corps (Medical).

The following officer is placed upon the Retired List (Eastern Command) with permission to retain his rank and wear the prescribed uniform, 31st August, 1959.

Captain K. G. Lawrence.

ROYAL AUSTRALIAN AIR FORCE.

Air Force Reserve.

Medical Branch.

The following officers are promoted to the temporary rank of Wing Commander, 27th August, 1959:—Flight Lieutenants (temporary Squadron Leaders) W. A. Dott (257935), H. P. Greenberg (267761), N. Y. McCallum (277624), C. F. McCann (257631), N. Morrissy (254850), Flight Lieutenant (acting Squadron Leader) L. E. McDonnell (267717).

Flight Lieutenant W. W. J. Liddle (016466) is promoted to the temporary rank of Squadron Leader, 27th August, 1959.

The provisional appointment of Pilot Officer J. R. Casley-Smith (042358) is confirmed and he is promoted to the rank of Flight Lieutenant, 30th March, 1959.

Flying Officer W. G. Boxall (16440) is promoted to the rank of Flight Lieutenant, 1st October, 1959.

Flight Lieutenant W. G. Boxall (16440) is transferred from the General Duties Branch, 1st October, 1959.

The appointment of the following officers is terminated, 30th September, 1959:—Wing Commander M. J. H.

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED DECEMBER 19, 1959.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.	Australian Capital Territory.	Australia.
Acute Rheumatism	3(1)	1	2(1)	1	..	..	..	..	7
Amœbiasis	..	..	..	..	..	..	..	..	..
Ancylostomiasis	..	..	..	..	..	..	8	..	8
Anthrax	..	..	..	..	..	..	..	..	..
Bilharziasis	..	..	..	..	..	..	..	..	..
Brucellosis	1	..	..	..	..	..	..	..	1
Cholera	..	..	..	..	..	..	..	..	..
Chorea (St. Vitus)	..	..	..	..	..	..	..	..	..
Dengue	..	..	..	..	..	..	..	..	..
Diarrhœa (Infantile)	9(5)	5(5)	1(1)	..	2	..	2	..	19
Diphtheria	..	..	..	..	..	..	..	..	..
Dysentery (Bacillary)	..	..	2	..	..	..	..	..	2
Encephalitis	1	..	..	..	..	..	..	..	1
Filariasis	..	..	..	..	..	..	..	..	..
Homologous Serum Jaundice	..	..	..	..	..	..	..	..	..
Hydatid	..	..	..	..	..	..	..	..	..
Infective Hepatitis	68(21)	15(10)	14(4)	26(14)	3(2)	1(1)	..	1	128
Lead Poisoning	..	..	2	..	..	..	..	..	2
Leprosy	..	..	..	..	..	..	..	..	..
Leptospirosis	..	..	5	..	..	..	..	..	5
Malaria	..	..	..	..	..	..	..	..	..
Meningococcal Infection	..	4(1)	1	..	..	1(1)	1	..	7
Ophthalmia	..	..	..	1	5	..	1	..	7
Ornithosis	..	..	..	..	..	..	..	..	..
Paratyphoid	..	..	..	..	..	..	..	..	..
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	..	..	1	..	..	..	..	..	1
Puerperal Fever	..	..	..	..	..	..	..	..	..
Rubella	..	7(3)	..	2	2(2)	..	..	2	13
Salmonella Infection	..	..	..	8(6)	2	..	..	..	10
Scarlet Fever	6(3)	13(9)	2(1)	4(4)	2(2)	1(1)	..	..	28
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	..	..	1	..	..	..	..	..	1
Trachoma	..	..	..	..	14	..	18	..	32
Trichinosis	..	..	..	..	..	..	..	..	..
Tuberculosis	13(6)	10(5)	6(4)	7(4)	13(6)	3(1)	2	..	54
Typhoid Fever	..	..	..	..	..	..	..	..	..
Typhus (Flea-, Mite- and Tick-borne)	..	..	1	..	..	..	..	..	1
Typhus (Louse-borne)	..	..	..	..	..	..	..	..	..
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] Figures in parentheses are those for the metropolitan area.

Hutchison (261805); Flight Lieutenants (temporary Wing Commanders) H. C. Finn (2079), W. N. Little (6410), B. T. Mayes, M.V.O. (264848), A. S. DeB. Cocks (281255); Flight Lieutenants (temporary Squadron Leaders) H. C. Taylor (264368), L. H. Albiston (256799), J. S. Bothroyd (251461), R. M. Cloutier (264595), W. T. Coyle (261680), A. E. Dickmann (252396), E. J. Egan (268061), J. D. Holmes (292095), G. Matthews (263561), W. T. D. Maxwell (264363), Flight-Lieutenants J. T. Broughton (267513), C. B. Carver (287423), L. Champion (257719), B. R. Cooke (1478), E. L. Davey (251236), J. D. G. Dunn (251233), F. G. Fenton (257728), A. W. Graham (3954), R. J. C. Kristenson (264418), T. B. Law (2566), H. B. Little (3452), T. P. Mahon (261455), H. C. Maling (1479), A. J. May (277567), F. C. Middleton (3451), J. M. C. Philpott (1470), J. L. Sertori (267565), J. S. Steel (267535).

Notice.

THE CHILDREN'S MEDICAL RESEARCH FOUNDATION OF N.S.W.

THE following is a list of donations to the Children's Medical Research Foundation of N.S.W. received from members of the medical profession in the period December 10, 1959, to January 9, 1960.

Dr. N. A. Keirle and Mrs. E. A. Keirle, £100.

Dr. Verlie Lines, £20.

Dr. David Garrett (further), Dr. B. A. Thomas, Dr. A. Pfeifer, Dr. H. B. Cribb (further), £5 5s.

Dr. C. Denys Garnsey, Dr. E. P. Blashki (further), £5.

Dr. S. Rosenberg (further), £2 2s.

Previously acknowledged: £9998 12s. 10d. Total received to date: £10,151 14s. 10d.

Australian Medical Board Proceedings.

NEW SOUTH WALES.

THE following additions and amendments have been made to the Register of Medical Practitioners for New South Wales, in accordance with the provisions of the *Medical Practitioners Act*, 1938-1958.

Registered medical practitioners who have complied with the requirements of Section 17 (3) and are registered under Section 17 (1) (a) of the Act: Morrison, Rodney Robert Winston, M.B., Ch.B., 1952 (Univ. New Zealand), D.P.H. (New Zealand), 1956; Pilkington, Reginald Ralph, M.B., B.S., 1937 (Univ. Melbourne).

Registered medical practitioners who have complied with the requirements of Section 17 (3) and are registered under Section 17 (1) (b) of the Act: Cohen, Maurice, M.B., B.S., 1952 (Univ. London); Duguid, Barbara, M.B., B.S., 1951 (Univ. London); Edmiston, Donald Fleming, M.R.C.S., England, L.R.C.P., London, 1942; McCarthy, John Hackett, M.B., B.Ch., 1954 (N.U. Ireland); McIlrath, William Arthur, M.B., B.Ch., 1928 (Q.U. Belfast), D.P.M., R.C.P. & S., Ireland, 1950, D.P.M., London, 1950; Murugasu, James Jayapalan, M.B., B.S., 1953 (Univ. Malaya); Thevathasan, Christopher Gnanaratnam, M.B., B.S., 1954 (Univ. Malaya); Weaver, George Alfred, M.B., B.Ch., 1955 (Univ. Dublin).

Registered medical practitioner who has complied with the requirements of Section 17 (3) and is registered under Section 17 (2A) of the Act: Papaharalambous, Athanasios, M.D., 1940 (Univ. Athens).

The following have been issued with a licence according to Section 21A of the Act: Gerlach, Helmut, Ivanhoe Region, one year from December 1, 1959; Frommer, John Eugene, Cassilis Region, one year from December 1, 1959; Csillag, John, Ungarie Region, one year from December 1, 1959.

Nominations and Elections.

THE following have applied for election as members of the New South Wales Branch of the British Medical Association:

Coupland, Graham Arthur Edwin, M.B., B.S., 1959 (Univ. Sydney), 27 Anthony Road, West Ryde, New South Wales.

Burgess, John Austin, M.B., B.S., 1959 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown, N.S.W.

Deaths.

THE following deaths have been announced:

BROWN.—Gilbert Brown, on January 7, 1960, at Adelaide.

PFEIFFER.—Gordon Harold Pfeiffer, on January 8, 1960, at Sydney.

GODFREY.—Graham George Godfrey, on January 10, 1960, at Melbourne.

Diary for the Month.

JANUARY 26.—New South Wales Branch, B.M.A.: Medical Politics Committee.
JANUARY 26.—Tasmanian Branch, B.M.A.: Southern Subdivision.
JANUARY 27.—Victorian Branch, B.M.A.: Branch Council.
JANUARY 28.—Tasmanian Branch, B.M.A.: Northern Subdivision.
FEBRUARY 2.—New South Wales Branch, B.M.A.: Organisation and Science Committee.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales.

South Australian Branch (Honorary Secretary, 80 Brougham Place, North Adelaide): All contract practice appointments in South Australia.

Editorial Notices.

ALL articles submitted for publication in this Journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations, other than those normally used by the Journal, and not to underline either words or phrases.

Authors of papers are asked to state for inclusion in the title their principal qualifications as well as their relevant appointment and/or the unit, hospital or department from which the paper comes.

References to articles and books should be carefully checked. In a reference to an article in a journal the following information should be given: surname of author, initials of author, year, full title of article, name of journal, volume, number of first page of article. In a reference to a book the following information should be given: surname of author, initials of author, year of publication, full title of book, publisher, place of publication, page number (where relevant). The abbreviations used for the titles of journals are those of the list known as "World Medical Periodicals" (published by the World Medical Association). If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors submitting illustrations are asked, if possible, to provide the originals (not photographic copies) of line drawings, graphs and diagrams, and prints from the original negatives of photomicrographs. Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary is stated.

All communications should be addressed to the Editor, THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2-3.)

Members and subscribers are requested to notify the Manager, THE MEDICAL JOURNAL OF AUSTRALIA, Seamer Street, Glebe, New South Wales, without delay, of any irregularity in the delivery of this Journal. The management cannot accept any responsibility or recognize any claim arising out of non-receipt of journals unless such notification is received within one month.

SUBSCRIPTION RATES.—Medical students and others not receiving THE MEDICAL JOURNAL OF AUSTRALIA in virtue of membership of the Branches of the British Medical Association in Australia can become subscribers to the Journal by applying to the Manager or through the usual agents and booksellers. Subscriptions can commence at the beginning of any quarter and are renewable on December 31. The rate is £6 *per annum* within Australia and the British Commonwealth of Nations, and £7 10s. *per annum* within America and foreign countries, payable in advance.